PERINATAL OUTCOME IN TWIN PREGNANCY ACCORDING TO CHORIONICITY

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CERTIFICATE

This is to certify that the dissertation entitled "**PERINATAL OUTCOME IN TWIN PREGNANCY ACCORDING TO CHORIONICITY"** is a bonafide work done by **Dr.G.ARUNADEVI** in the Institute of Social Obstetrics, Govt Kasturba Gandhi hospital (Madras Medical College) Triplicane , Chennai, in partial fulfillment of the university rules and regulations for award of MD degree in Obstetrics and Gynaecology under my guidance and supervision during the academic year 2012-2014.

Prof. DR.V.KANAGASABAI M.D

The Dean,

Rajiv Gandhi Govt. general hospital,

Madras Medial College,

Chennai-3.

Prof. DR.S.DILSHATH M.D, D.G.O

Director,

Institute of Social Obstetrics,

Government Kasturba Gandhi Hospital,

Chennai – 3.

Prof. DR.B.TAMILSELVI, M.D., DGO.

Guide

Professor,

Institute of Social Obstetrics,

Govt. Kasturba Gandhi hospital, Chennai-3

DECLARATION

I solemnly declare that this dissertation entitled **"PERINATAL OUTCOME IN TWIN PREGNANCY ACCORDING TO CHORIONICITY"** was done by me at The Institute of Social Obstetrics, Govt Kasturba Gandhi Hospital, Madras Medical College during 2012-2014 under the guidance and supervision of, **Prof. Dr. B. TAMILSELVI MD, DGO.** This dissertation is submitted to the TamilNadu Dr. M.G.R. Medical University towards the partial fulfillment of requirements for the award of M.D Degree in Obstetrics and Gynaecology (Branch-II).

Place: Chennai Signature of Candidate

Date:

Prof. DR.B.TAMILSELVI.M.D., DGO.

Guide

Institute Of Social Obstetrics,
Govt.Kasturba Gandhi Hospital
Madras Medical College
Chennai-3.

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CONTENTS

LIST OF ABBREVIATIONS

APH	Ante partum haemorrhage
CHD	Congenital heart disease
CTEV	Congenital talipes equino varus
DC	Dichorionic
DCDA	Dichorionic diamniotic
FSH	Follicle Stimulating Hormone
GDM	Gestational diabetes mellitus
IUD	Intra Uterine Death

IUFD	Intra uterine fetal death
IUGR	Intra uterine growth retardation
IUI	Intra uterine insemination
IVF	In Vitro Fertilization
IVH	Intra ventricular haemorrhage
LBW	Low birth weight
LSCS	Lower segment caesarian section
MC	Monochorionic
MCDA	Monochorionic diamniotic
MCMA	Monochorionic monoamniotic
NEC	Necrotising enterocolitis
NICU	Neonatal intensive care unit
PIH	Pregnancy induced hypertension
PPH	Post partum haemorrhage

PPROM	Preterm premature rupture of membrane
PROM	Preterm rupture of membrane
RDS	Respiratory distress syndrome
TAPS	Twin anemia polycythemia sequence
TRAP	Twin reversed arterial perfusion
TTTS	Twin to twin transfusion syndrome
VLBW	Very low birth weight
VxVx	Vertex vertex

PERINATAL OUTCOME IN TWIN PREGNANCY ACCORDING TO CHORIONICITY

INTRODUCTION

Twin pregnancies have been increasing in incidence over a few decades. Use of ovulation induction with drugs, in vitro fertilization and increasing age of the mother during conception are two primary causes for the increase in incidence[1].

Twin pregnancies though accounting for only a lesser percentage for live births, are known to account for a disproportionate percentage for all the adverse perinatal outcomes. The major problems occurring in twin pregnancy are prematurity, low birth weight, intra uterine growth retardation, birth trauma, birth asphyxia and congenital anomalies and fetal complications peculiar to twin pregnancies. About one fourth of twins require neonatal (NICU) admission. Twins when compared to singleton pregnancy, have a fivefold risk of dying before they reach one year. Mother of a twin pregnancy has a risk of getting transferred to ICU at a rate of 3.1%, whereas for a singleton pregnancy it is only 0.3%[2]. Because of the risks involved in twin pregnancies, they demand extremely vigilant antepartum, intrapartum and postpartum care.

Twins can be either monozygotic or dizygotic. Dizygotic twins or fraternal twins are formed due to fusion of two separate ova by two different sperms. The placenta is always dichorionic and diamniotic. Depending on the timing of splitting of fertilized egg, monozygotic twins can be dichorionic or monochorionic. When the splitting occurs within 3 days of fertilization it results in the formation of dichorionic twins and when it occurs after 3 days, monochorionic twins are formed.

The perinatal complications in MC twin pregnancies are higher than DC twins[3-6]. The reason for such an increase is that MC twins have a shared placenta with vascular anastomoses which in turn leads to shunting of blood between the two twins. Twin-to-twin transfusion syndrome (TTTS) which develops in 10-15% of MC twin pregnancies has a grave prognosis if proper treatment is not given. If one twin of a MC pregnancy dies and the other one survives, the surviving twin because of vascular anastomoses has a still higher perinatal morbidity and mortality when compared with DC twins[7]. The problems faced by surviving twins are very distressing like cerebral impairment or preterm delivery and its sequelae[7, 8].

Even if antenatally diagnosed TTTS is excluded, mortality of MC twins is still more when compared to DC twins. There are also reports of late fetal deaths In MC twins, that is, deaths occurring after 32 weeks of gestation. Various studies give variable death rates for late fetal deaths, thus the decision regarding time to deliver MC twins is controversial. Even if there is no TTTS or IUD of co-twin, neonatal morbidity is still more in MC twins.

Hence I have undertaken this thesis to analyse the perinatal morbidity and mortality in twin pregnancy with relation to chorionicity so as to use the knowledge of maternal and perinatal complication in twin pregnancy for better maternal surveillance and in prevention and treatment of the complications. This will improve the perinatal and maternal outcome.

AIMS AND OBJECTIVES OF THE STUDY

- To study the perinatal morbidity & mortality in twin gestations according to chorionicity.
- To study the causes for adverse perinatal outcome.
- To study the number of babies discharged without risk factors.
- To evaluate and compare complications in monochorionic and dichorionic twin gestations.

REVIEW OF LITERATURE

Twin pregnancies have been increasing in incidence all over the world and in India. Perinatal morbidity and mortality are also higher in twin pregnancies. Prematurity and low birth weight are the two major causes for the perinatal morbidity and mortality. Other causes in the list are fetal malpresentations and complications associated with labour. These issues make twin gestation a high risk gestation. Twin pregnancies are always a major cause of concern and the mode by which they are delivered is a problem to the obstetricians[9].

Twins may be identical (monozygotic) or non-identical and fraternal (dizygotic).

Dizygotic twins:

These are the common type of twins and constitute for two thirds of twin pregnancies. Increasing maternal age increases the incidence of these twins[10]. They are formed as a result of fusion of two ova by two separate sperms. The genetic composition of the two zygotes are different. Each zygote develops its own placenta, amnion and chorionic sac. Dizygotic twinning depends on conditions like age of the mother, race to which she belongs, preconceptional weight, obstetric history and the time of year of the conception. The levels of

gonadotropic hormones in maternal blood of dizygotic twins is found to be increased[9].

Monozygotic Twins:

These are twins which develop from a single ovum and are identical in nature. The frequency of monozygotic twins is one in 250 pregnancies[9]. This twinning occurs as a random genetic event. There are 3 types of monozygotic twin pregnancies depending on the timing of division of the zygote. The three types are as follows:

Dichorionic diamniotic: when the splitting occurs within 4 days of fertilization.

Monochorionic diamniotic: If the splitting tends to occur between the 4 th day to 7 th day of fertilization.

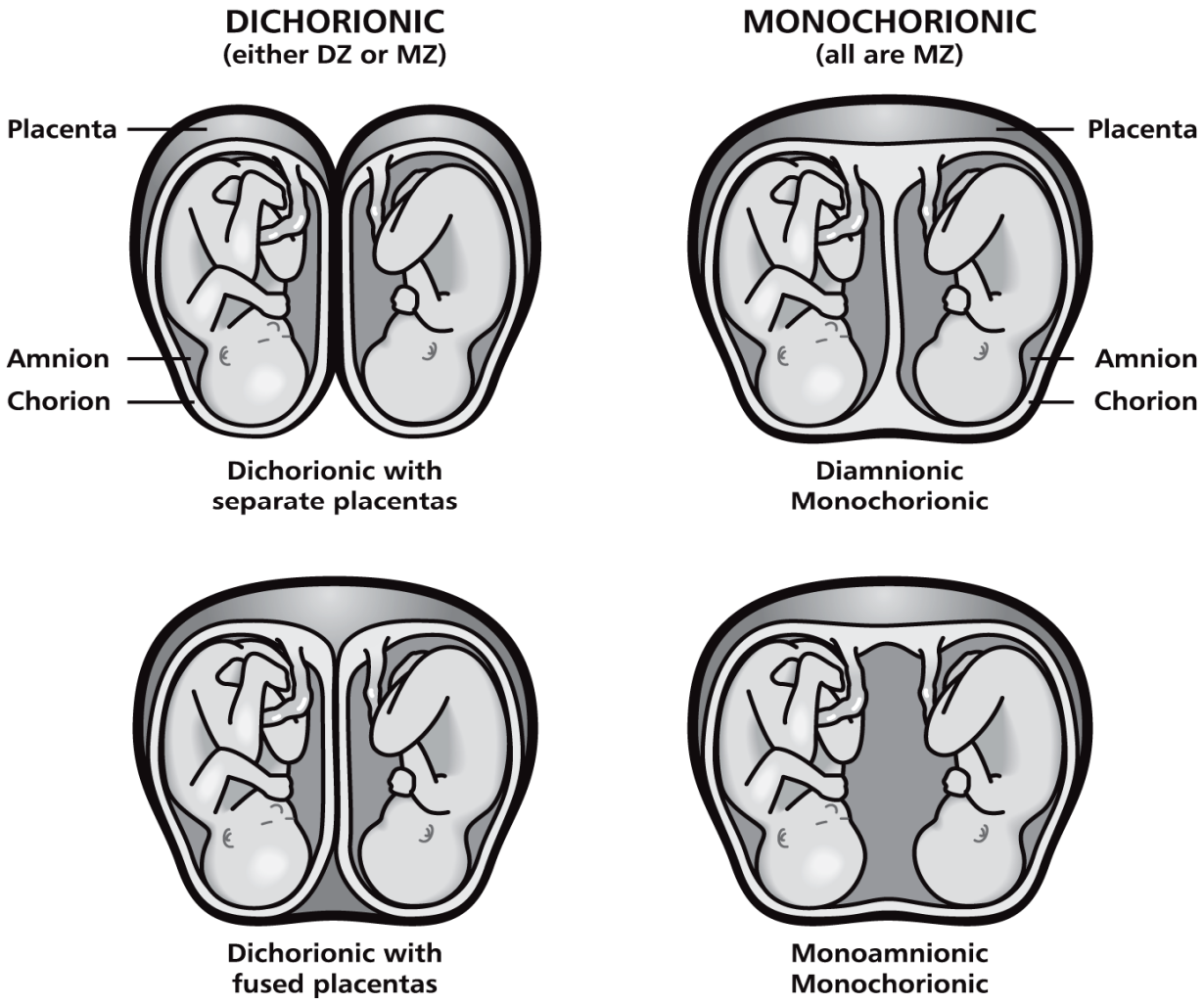
Monochorionic monoamniotic: If it occurs > than 7 days after fertilization.

Conjoined twins: When the twinning event occurs late at around 13 to 14 days after the event of fertilization conjoined twins are formed.

In contrast to dizygotic pregnancies, monozygotic pregnancies are not affected by factors like age of the mother, her parity, her ethnicity, nutritional factors and environmental conditions. However the usage of assisted reproductive

technologies like ovulation induction and in vitro fertilization increases the incidence.

Figure showing amnionicity and chorionicity



PHYSIOLOGIC ADAPTATIONS OF THE MOTHER TO MULTIPLE GESTATION:

As a result of multiple pregnancy the normal ability of the woman to adapt herself to the needs of pregnancy are significantly altered[11]. The knowledge about maternal changes in singleton pregnancy and those specific to twin pregnancy is essential for the treating doctors to predict the complications specific to twinning and manage them effectively.

Cardiovascular System:

In a singleton pregnancy, the plasma volume of the mother increases by 10% at the 7th week and increases by 45 to 50% by 32 weeks and becomes constant thereafter. Study by Pritchard has shown that the average blood volume increase in a singleton pregnancy is 1570 ml compared to twin pregnancy where it is 1960 ml[12].

Respiratory system:

During a normal singleton pregnancy, several changes occur in the maternal respiratory system. Functional residual capacity reduces as a result of elevation of the relaxed diaphragm. The enlarging uterus which enters the abdominal cavity causes an increase in inspiratory reserve volume. Increase in the anteroposterior

diameter of the thoracic cage and increased flaring of ribs compensates for the reduction in residual volume, total lung capacity and expiratory reserve volume[13]. Information regarding the changes in respiratory system in multiple pregnancy are unknown. Except for the elevation of diaphragm to a greater extent, no other change differentiates multiple from singleton pregnancy with regard to the respiratory system.

Renal System:

Glomerular filtration rate in a singleton pregnancy increases by around 50% by the time of 12 weeks. It reaches a peak of approximately around 180 ml/min from the normal value of 120 ml/min. No significant difference in GFR is noted between twin and singleton gestation with regard to GFR[14].

Gastrointestinal system:

Progesterone levels are increased in twin gestations. This causes changes in the functioning of digestive and gastrointestinal system. Progesterone, in addition to decreasing the peristalsis of intestines and stomach, also decreases the lower esophageal sphincter tone. Food absorption is also decreased. The intraabdominal part of the esophagus is displaced by the enlarging uterus into the

thoracic cage and this decreases the tone of the lower osophageal sphincter which usually protects the esophagus from reflux gastric juice rich in acid and pepsin[9].

Hematologic system:

Pregnancy is a potential thrombogenic state. Fibrinogen, factors XII, X, IX, VII, VIII and von Willebrand factor levels increase in the blood. Factor XI falls and prothrombin and factor V remain the same. Anticogulants like protein C and antithrombin III levels also falls or remain the same and protein S falls[15]. As a result of these changes there is an increased susceptibility towards thrombosis in antenatal period and till 6 weeks postpartum. These changes mainly help in bringing down the loss of blood during delivery.

INCIDENCE AND FACTORS AFFECTING TWINNING:

Monozygotic twins have a constant rate of prevalence all over the world. The prevalence rate is between 0.3 and 0.4%. Mammals except armadillos have the same rate of prevalence. History of twinning in a female does not alter the subsequent risks for twinning.

However the prevalence of dizygotic twinning is not constant and it varies between different populations. In some areas the prevalence rate is as low as 0.6% whereas in other areas it is around 4.5%. This difference is because of various factors like age of the mother, race, parity etc. which influence the twinning rate in dizygotic pregnancies.

Race:

The levels of Follicle stimulating hormone has been found to vary among various populations. Because of this, there is a racial difference in twinning rate[16].

Heredity:

Presence of family history of twinning on the paternal side positively influences the incidence of twinning[16].

Maternal age and parity:

At the age of 37 years, FSH stimulation is at its maximum causing the development of multiple follicles. Hence the rate of twinning is also maximum at this age. After 37 years, the follicles are depleted and hence the rate of twinning

decreases. Twinning also increases as the parity becomes more in all groups of people[16].

Nutritional factors:

Women with good nutritional status like those who are tall and heavy had a 25 to 30% higher incidence of twinning when compared to women who are small. This phenomenon was confirmed from the study by Macgillivray et al who also found that dizygotic twinning is more common in large and tall women when compared to small women[16].

Pituitary Gonadotropin:

Benirschke and Kim in 1973 suggested that FSH level may be the common factor which links age, weight, race and fertility to multiple gestation.

Therapy for infertility:

The usage of assisted reproductive techniques has been increasing. Reports suggest that induction of ovulation with clomiphene citrate and FSH along with chorionic gonadotropin increases the probability of having multiple ovulations. After taking gonadotropin therapy, the incidence for having a multiple pregnancy

is 16 to 40%. Out of this, 75% is constituted by twins. When greater number of embryos are transferred in IVF, the risk of getting multiple fetuses and twins increase[16].

DIAGNOSIS OF CHORIONICITY BY SONOGRAPHY AND PLACENTAL EXAMINATION:

Because the perinatal outcome in MC pregnancies is poor when compared to DC pregnancies, the assessment of chorionicity becomes essential. Assessment of chorionicity assists in the optimal management of pregnancy and its complications. For example when there is an IUGR, knowledge of chorionicity is helpful to decide whether it is caused by uteroplacental insufficiency or by TTTS.

Information about chorionicity is also useful when doing elective first trimester multifetal pregnancy reduction (MPR) or when selective termination (ST) of one abnormal fetus is planned. When selective termination (ST) of one abnormal twin is done in MC pregnancies, as a result of placental sharing, death or injury to the surviving twin may occur.

Sonographic evaluation:

First trimester scan can diagnose chorionicity with precision. The presence of two separate placentas and a thick dividing membrane between the two placentas which is usually 2 mm thick is more in favour of dichorionicity. When there is a male and female fetus rather than both of same sex, it favours dizygosity and dichorionicity.

When there is a single placental mass there may be confusion whether it is a single large placenta or two small placentas lying side by side. In such cases, the point of origin of the dividing membrane is analysed carefully. The twin peak sign or (lambda) sign is said to be present when there is a triangular projection of placental tissue extending between the layers of the dividing membrane.

Twin peak sign

The dividing membrane of MC pregnancies is very thin, usually < 2 mm and is best made out in second trimester. Scardo et al in 1995 stated that only two layers are made out even with magnification. The 90^0 relationship between the placenta and membranes with no extension of placenta between them is called as the T sign. Stagiannis et al in 1995 stated that performing a first trimester ultrasound is the best for determining chorionicity, since the dividing membrane is easily evaluated at that time when the fetuses are small in size.

T sign

In 2006, Lee et al compared the efficacy of ultrasound and examination of placenta after delivery for determination of chorionicity. The parameters used in the ultrasound were placental location, fetal sex, whether twin peak sign is present or absent. A total of 410 twin pregnancies were analysed. It was found

that ultrasound diagnosis was 96% accurate in determining chorionicity. Scanning was more sensitive when done in the first trimester rather than in second[16].

RCOG also recommends that chorionicity should be best determined before 14 weeks[40]. As the gestatational age increases, the chorion frondosum regresses. Thus lambda sign is lost towards 20 weeks. Usage of high frequency ultrasound in the range of 7.5 to 10 MHz transducers gives better results. Using that frequency, the predictive value for dichorionicity was found to be 100% and 100% for monochorionicity[41].

Placental examination:

The placenta should be carefully examined for determination of chorionicity. It should be examined as follows:

Deliver one baby first and put a clamp on its umbilical cord. The cord blood should be collected only after the delivery of the second twin. If there are two separate placentas without any doubt, then cord blood may be collected before the delivery of second twin. Two clamps should be placed on the cord of second baby after its delivery and 3 clamps for the third baby and so on. The cords should be

clamped till the last baby is delivered. If not done, fetal anemia and hypovolumia would result because of blood leaving the placenta through anastomosis.

When the placenta is delivered, the attachmaent of chorion and amnion should be preserved because the relationship of membranes is essential in determining chorionicity. Monozygotic fetuses have one common amnionic sac or apposed amnions not separated by chorions. When the adjacent amnions are separated by chorion, the fetuses are usually dizygotic but can be monozygotic also.

Sometimes blood group determination of the fetuses also help in determining chorionicity. When the two fetuses have different blood group, it confirms the chorionicity as dizygotic whereas presence of same blood group does not confirm monozyosity. St. Clair and associates suggested that other tests like finger printing may be used for the diagnosis.

Study by Ingruise Louse et al showed that dizygotic twin pairs share two HLA haplotypes more commonly than ordinary siblings born out of separate pregnancies and are thus genetically more alike.

PERINATAL OUTCOME ACCORDING TO CHORIONICITY:

Studies by De Snoo et al between 1907 to 1938 conducted on 651 twin pregnancies in Rotterdam showed that perinatal mortality was higher in monochorionic pregnancies[18].

Ana patrica Domingue et al conducted a study on 323 twin pregnancies at Hospitais da Universidade de Coimbra between January 2000 and December 2005. It was inferred that complications were more frequent in monochorionic than in dichorionic pregnancies. Differences between the two groups were significant for fetal mortality, discordant growth, fetal growth restriction and preterm delivery[19].

Dias T et al conducted a study on entanglement of cord and the perinatal outcome in monoamniotic twin pregnancies. Totally thirty two monoamniotic pregnancies were included in the study. Entanglement of umbilical cord was seen in all monoamniotic fetuses when it was evaluated by sonography and color Doppler. Mortality in monoamniotic twins was due to conjoined twins, TRAP, and discordant fetal growth[20].

Nihal Al Riyami et al did a retrospective analysis of 51 twin pregnancies delivered at Sultan Qaboos University Hospital during January 2006 to December 2011. Thirty six (71%) pregnancies were dichorionic diamniotic (DCDA), 14 (27%) were monochorionic diamniotic (MCDA), and one (2%) was monochorionic monoamniotic (MCMA). Perinatal morbidity and mortality remained high among monochorionic twins. This was likely due to frequent prematurity, fetal growth restriction, TTTS, and fetal death[21] occurring in the intrauterine period.

Victoria A et al conducted a study on pregnancy outcome in perinatal period, pathology of the placenta, and severity of discordance in MC and DC twins. A cohort of 382 twin gestations with gestational ages from 24 to 40 weeks was studied retrospectively. Severe discordance was more rampant and found to have greater morbidity among MC than DC twins. Findings noted in the placentas of severely discordant twins were reduced weight of the placenta and cord abnormalities[22].

COMPLICATIONS OF TWIN PREGNANCY AND ITS RELATION TO AMNIONICITY AND CHORIONICITY:

Both maternal and fetal complications arise with twinning. Maternal complications are as follows:

Antenatal complications

Gestational diabetes:

The incidence of GDM increases in twins compared to singletons. 22 to 39% of triplets have gestational diabetes as compared to twins where the rate is 3 to 6%[23, 24].

Hypertension and preeclampsia:

Gestational hypertension is found to be more common in twin than in singleton pregnancies. Preeclampsisa is found to occur 2.6 times more commonly in twin than in singleton gestations and is even more common in triplet pregnancies[25, 27,28].

Anemia:

The incidence of anemia in twin pregnancies is 9.4% whereas in singleton gestations it is 4.1%. Multiple fetuses need extra iron for their growth and the red cell mass increases further thus increasing the incidence of anemia[41].

Antepartum hemorrhage:

Antepartum hemorrhage due to abruptio placenta and placenta previa are found to be twice as common in twin pregnancies as in singleton pregnancies[9].

Other pregnancy complications:

Acute fatty liver as a complication of pregnancy is rare. But when it occurs, it occurs disproportionately more commonly in twin gestations. It occurs one in 10,000 singleton pregnancies. It complicates 2% of pregnancies, out of which 14% occur in twin pregnancies and 7% in triplet pregnancies[29, 30].

Postnatal complications

Postpartum bleeding:

Uterine atony is very common following the delivery of twins. This causes an increase in the incidence of PPH. The uterine muscle fibers are stretched to the maximum when delivery occurs near term compared to delivery in earlier weeks. As a result PPH commonly occurs when delivery is near term[41]

Fetal complications:

Complications like premature delivery of one or both fetuses, discordant growth abnormalities, intrauterine demise of both or one fetus, preterm premature rupture of the membranes are the ones which are troublesome to the treating obstetrician and paediatrician. MC pregnancies have varied complications like twin to twin transfusion syndrome which altogether alters the management of multiple pregnancy. Multiple pregnancy in general has significant perinatal morbidity and mortality which warrants special consideration.

Complications of fetus in relation to chorionicity:

Vascular complications occur commonly in MC twins. The most dreaded vascular complication is twin to twin transfusion syndrome. It is found to occur in 10 – 15% of MC pregnancies. Other complications which can occur are growth restriction, twin anemia polycythemia sequence, congenital heart disease, twin reversed arterial perfusion, fetal death, and long-term morbidity secondary to vascular injury in fetal brain, heart and kidneys[31].

Twin-to-twin transfusion syndrome (TTTS):

TTTS occurs because of sharing of placenta and vascular anastomosis. Blood flow to the fetuses are not balanced as a result of which the hemodynamics and hormonal balance are altered[33]. Otherwise known as Oligohydramnios polyhydramnios sequence, it is present in around 15% of MC gestations. It tends to occur whatever be the mode of conception.

Untreated TTTS causes intra and perinatal death in 90% of cases. 50% of surviving twins has neurological sequelae as a result of prematurity or death of one twin[34]. TTTS can be diagnosed at any gestational age. Signs of development of TTTS in ultrasound are membrane folding, discordance in fetal growth between the two twins, increased thickness of nuchal translucency and increase in amniotic fluid volumes. When the mother develops acute symptoms related to polyhydramnios like distension of uterus, uterine contractions and dyspnea , TTTS should be a diagnostic consideration[33].

TTTS may also occur as a complication of monoamniotic twins. Since these placentas do not have a dividing membrane, oligohydramnios is unlikely to develop in the donor. Polyhydramnios is present in the single amniotic cavity.

Diagnosis can be made out through Doppler studies and the presence of polyhydramnios and difference in bladder filling helps in the diagnosis in such cases.

Twin reversed arterial perfusion (TRAP) sequence:

TRAP is a unique complication of MC gestations. The presence of arterio arterial anastomosis causes pumping of blood to the acardiac twin which is a true parasite. High output cardiac failure and prematurity secondary to polyhydramnios is the cause for death of the pump twin[42].

Twin anemia polycythemia sequence (TAPS):

Twin anemia polycythemia sequence is a variant of TTTS. Small unidirectional artery to vein anastomosis causes a raised hemoglobin level in one twin and chronic anemia with reticululocytosis in the other twin with no evidence of oligohydramnios-polyhydramnios sequence[35]. There occurs chronic net transfusion across the minute anastomosis. 5% of previously uncomplicated MC gestations can develop TRAP subsequently.

TRAP is treated by transfusing the anemic twin and by causing hemodilution to the polycythemic co – twin.

Selective intrauterine growth restriction:

Selective intrauterine growth restriction (sIUGR) accompanies MC pregnancy commonly. It secondarily causes intrauterine fetal death and neurological sequelae in both twins and hence is considered an important complication of MC pregnancies[36].

sIUGR is termed based on the estimated body weight of smaller fetus. When it falls below the 10 th percentile, then the term is used. Though most of the times,

weight discordance between the two twins accompanies this issue, weight discordance is not needed for its diagnosis.

sIUGR has a prevalence rate of 10 – 15%. When compared with TTTS, sIUGR has lesser mortality with a rate of 10% whereas in TTTS it is as high as 55%[37]. Potential cause for nightmare with such sIUGR twins is intrauterine death of the growth restricted twin. Even if the babies are born alive, there still exists a significant risk for the normally grown twin.

The placenta is not adequately shared between the two twins and this is found to be the reason for development of sIUGR. The presence of vascular anastomosis in MC placenta is the second reason for weight discordance and also the development of sIUGR in MC twins, the first reason being discordance in placental territory[37].

Placental insufficiency and inter twin vascular communications are found to be the causes for significant differences in the outcome of MC pregnancies with similar levels of fetal weight discordance.

Monoamniotic twins:

Monochorionic monoamniotic twins are not common and they account for 1% of all MZ twins[38]. These are formed as a result of division of the embryo occurring 8 days after the conception. They have a single amnion and a yolk sac. They also may have two or one conjoined twin embryos.

Conjoined twins:

When the twinning event occurs late at around 13 to 14 days after the event of fertilization conjoined twins are formed. The splitting of the embryonal axis is not complete. They are classified into different types based on the region where their bodies are united. Management of such twins depends on ultrasound and fetoscopic assessment of fetal sharing.

Monoamniotic twins with cord entanglement:

The increased rate of perinatal mortality in monoamniotic twins is because of complications like cord entanglement, discordance of fetal growth, IUGR, twin to twin transfusion syndromes. Entanglement of the cord leading on to cord occlusion and fetal death is unique a complication of monoamniotic gestations. This problem complicates most of the monoamniotic pregnancies resulting in the

demise of one or dual fetuses. Color Doppler and antenatal sonography reliably detects cord entanlement[39].

PERINATAL OUTCOME IN TWIN PREGNANCY:

Preterm birth occuring in 44% of twins is the major contributor to the increased perinatal mortality rate in multiple gestations. Discordance of fetal growth is one of the most common complications of twinning. It may be a marker of placental insufficiency, twin-twin transfusion, genetic or structural anomalies. Low birth weight, congenital malformations, single intra uterine demise and still births are other potential complications of twin pregnancy.

Preterm delivery:

Twins when compared to singletons are more likely to be delivered preterm (< 37 weeks of gestation). In 2006, in the United States, approximately 60% of the twins were preterm and weighed less than 2500g. Approximately 1 out of 10 twin was born at below 32 weeks of gestation or weighed less than 1500g[31]. Overdistension

of the uterus and intra uterine infections are the important causes for preterm birth.

Comorbidity factors such as socioeconomical and ethnical factors, gestational age, premature rupture of membranes, fetal gender and availability of antenatal management with corticosteroids determine the perinatal mortality in twins.

The most frequent neonatal complications in preterm birth are respiratory distress syndrome, infections, hypothermia, persistent ductus arteriosus, intracranial bleeding, hypoglycemia, retinopathy of prematurity and necrotizing enterocolitis[41].

Preterm premature rupture of membranes:

The incidence of PPROM in multiple pregnancy (7.4%) is twice than that in singleton pregnancy (3.7%)[44]. Rupture takes place in the sac of presentation in majority of cases. The risk of infection, abruption, cord accident, overall perinatal mortality is the same in PPROM in twin as well as singletons.

Intrauterine Fetal Demise:

IUFD is precipated by several factors like multiple gestation, very low and very high age of the mother, postdated pregnancy, male sex of the fetus, and macrosomia of the fetus. The death of one twin occurs approximately in 3 – 4% of all twin gestations and the incidence is higher in monochorionic than in dichorionic gestations with a value of 26% in MC and 2.4% in DC[41].

Depending on the gestational age at which one twin dies, the outcome of the surviving twin is subject to vary. When one of the twins die before 14 weeks, there is no adverse outcome for the surviving twin. However, when one twin dies after 14 weeks, the other twin may also die or have severe morbidity.

When one twin dies, due to lack of blood pressure on that part of the circulation, massive shifting of blood occurs from the twin which survives to the twin which dies, thereby causing severe anemia in the surviving twin detected by Doppler of middle cerebral artery.

Birth weight:

As a result of restricted fetal growth and preterm delivery, the birth weight of multiple gestations is lower than singleton pregnancies[16].

Discordant growth:

Fetal weight discordance is defined as the difference between the estimated fetal weight of two fetuses as > 25% and is estimated by the subtracting the estimated fetal weight of the smaller twin from the estimated fetal weight of the larger twin and dividing it by the estimated fetal weight of the larger twin.

The perinatal mortality increases as the weight difference between the pair increases. In monochorionic twins, the difference is due to vascular anastomosis in the placenta which causes hemodynamic imbalance between the twins. Rarely discordancy for structural anomalies causes discordancy in weight in MC pregnancies.

Discordancy in dichorionic twins is due to varied reasons. Both the fetuses have different growth potentials in dizygotic twins and this could be the cause. Separate placentas require more implantation space and hence one of the placenta would have sub optimal implantation site and reduced growth of the corresponding fetus. So overcrowding of the uterus is found to play a role in growth discrepancies[16].

Congenital malformations:

The incidence of anomalies is significantly increased in multifetal pregnancies when compared to singleton pregnancies and especially in monozygotic pregnancies and it was 10.6% according to Hendricks et al. Anomalies arising in monozygotic pregnancies could be because of the process of twinning per se like conjoined twinning, acardiac anomaly and neural tube defects or due to vascular interchange in MC twins like microcephaly, intestinal atresia, aplasia cutis etc. or from fetal crowding like CTEV, congenital hip dislocation[43].

ROLE OF ASSESSMENT OF CHORIONICITY IN REDUCING ADVERSE PERINATAL OUTCOMES IN TWIN PREGNANCY:

Determination of chorionicity is important for early identification of complications of twinning and its prompt management.

Assessment of chorionicity is helpful in the following ways:

1. For Monochorionic twins**,** screening for growth discordancy and TTTS by ultrasound every two weeks from 16 weeks to delivery to be done.

2. For uncomplicated dichorionic, diamniotic twins, screening for growth at 28, 32 and 36 weeks of gestation to be done.

3. In MC pregnancies, usage of doppler ultrasound helps in diagnosing complications like growth discordance, IUGR, TTTS, TRAP and cord entanglement.

4. Chart growth for each fetus should be made at each scan to determine interval growth and overall growth velocity.

5. Structural defects are 2-3 times more common in live born MC twins than in DC twins. So advanced cardiac screening with fetal echo for all MC twins at 22-24 weeks is recommended.

6. Timing of delivery and its management should be planned based on chorionicity:

a. Fetal lung maturity occurs at an earlier gestational age in multiple gestations (32 weeks) compared to singleton gestations and hence term gestation is considered earlier in multiple than in singleton pregnancies. However, recommendation for delivery is at 37-38 weeks in uncomplicated dichorionic diamniotic (DCDA) twins or around 36 weeks in uncomplicated monochorionic diamniotic (MCDA) twins due to the increased risk of stillbirth.

b. Antenatal consultation with Newborn Care team and Neonatal Intensive Care Unit should be given, if birth planned at less than 36 weeks of gestation.

c. Consideration of antenatal steroids should be given for delivery prior to 34 weeks of gestation as per ACOG 2004 guidelines.

d. Women with an uncomplicated DCDA twin pregnancy presenting cephalic/cephalic or cephalic/non cephalic should be offered a vaginal birth.

Preterm birth occuring in 44% of twins is the major contributor to the increased perinatal mortality rate in multiple gestations. Discordance in fetal growth is a common complication of twinning. It may be a marker of placental insufficiency, twin-twin transfusion, genetic or structural anomalies. Evidence of fetal growth restriction, rather than discordance per se, predicts adverse neonatal outcome.

Serial scans throughout pregnancy are recommended given the inadequacy of clinical assessment of growth in multiple pregnancies.

Failure of proper surveillance of twin pregnancies leads to

a. Failure to diagnose correct chorionicity.

b. Failure to diagnose growth restriction and TTTS.

c. Failure to administer steroids to prevent neonatal respiratory distress.

Hence this thesis is being done to find out the perinatal morbidity and mortality of twin gestations according to chorionicity so as to assess the complications and extrapolate the data for offering best services to patients.

MATERIALS AND METHODS

Study centre : Govt. Kasturba Gandhi Hospital, ISO Chennai – 3

Duration of the study : 1 year between December 2012 to November 2013

Study design : Prospective study

Methadology (Materials and methods)

Inclusion criteria : Patients with twin pregnancy attending the antenatal OP and AN ward, labour ward of more than 28 weeks of gestational age.

Exclusion criteria : Patients with triplet pregnancy.

Patients with gestational age less than 28 weeks.

Patients with known H/O of chronic HT, DM, Chronic renal disease and other chronic medical disorders.

Sample size **:** 100

Method of data collection:

The information pertaining to the study like age, parity, gravida, residence, family history of twin pregnancy was obtained from the patients. Chorionicity was assessed using ultrasound and placental examination described earlier.

The perinatal outcome was recorded in terms of gestational age at delivery (28 – 30 weeks, 31-33, 34-37, > 37 weeks), mode of delivery (Caesarian section/vaginal delivery/combined/outlet forceps/vacuum), Apgar score at 0 and 5 mins, birth weight(> 2500 gms, 2500 – 1500 gms, < 1500 gms), gender, dead/still/alive, babies getting admitted to NICU, number of days in ICU, and the final outcome of the babies, in terms of whether the baby got discharged in good condition or expired.

Neonatal morbidity were further defined based on the causes like septicaemia, growth restriction, respiratory distress syndrome, septicemia, fetal growth

restriction (FGR), neonatal hyperbilirubinemia (NNH), patent ductus arteriosus (PDA), hypoglycemia, anomalous baby, neonatal seizures (NNS).

Causes of death were termed as due to Birth Asphyxia, Sepsis, Cord prolapse, Prematurity & its complications, Anomalous baby, Fetal growth restriction, neonatal seizures, intra uterine death.

The perinatal loss was defined as Intrauterine death or Neonatal death (≤28 days of birth) having a birth weight of >1 kg. Still births were also included in perinatal mortality. Stillbirth was Intra uterine death of a fetus weighing >1 kg and/or ≥28 weeks of gestation. Stillbirth was divided as ante partum deaths, where the fetuses had died before the start of labour, and intrapartum fetal deaths, where the fetuses had been alive at the onset of labour.

Perinatal morbidity was defined as 5-minute Apgarscore <7. Preterm confinement has been described as those delivered earlier to 37 weeks gestational age and very preterm birth has been described as those delivered earlier to 32 weeks. Low birth weight was defined as birth weight less than 2.5kgs and those weighing less than 1.5 kg as very low birth weight. A 5 minute Apgar score <7 was defined as a criterion of immediate neonatal morbidity. A 5-minute Apgar score <5 was considered as asphyxia.

Maternal conditions during antenatal period that affects perinatal outcome like PIH, GDM, APH, anemia complicating pregnancy were also studied.

Determination of chorionicity was done using sonography during pregnancy and by clinical assessment of placenta during delivery and described as dichorionic, monochorionic, diamniotic, monochorionic monoamniotic.

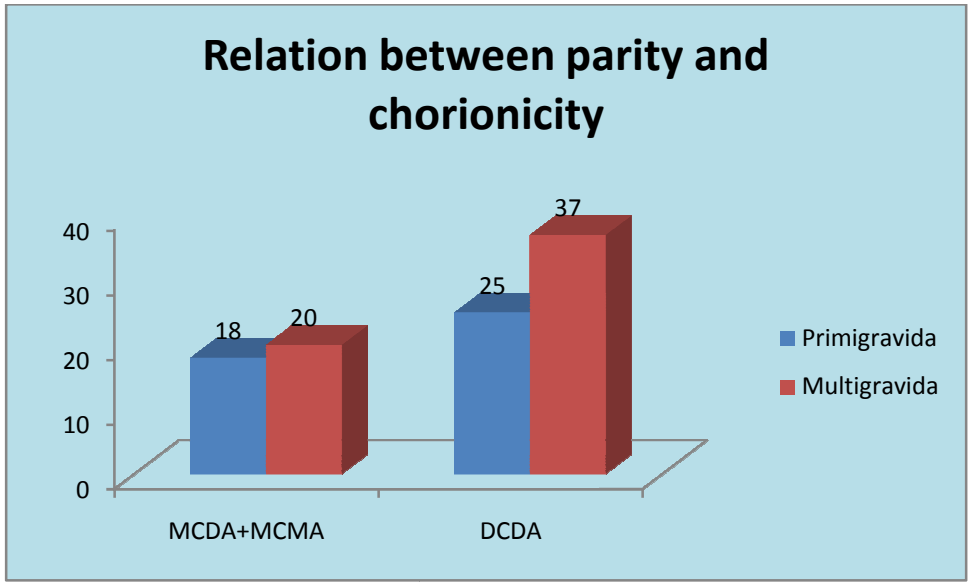
Congenital malformation was determined by sonography during pregnancy as well as by careful examination of the newborn baby.

RESULTS

Table 1 showing distribution of 100 twin deliveries with relation to parity & chorionicity:

Gravida	MCDA+MCMA	DCDA
Primigravida	18	25
Multigravida	20	37
Total	38	62

Figure 1 showing distribution of 100 twin deliveries with relation to parity:

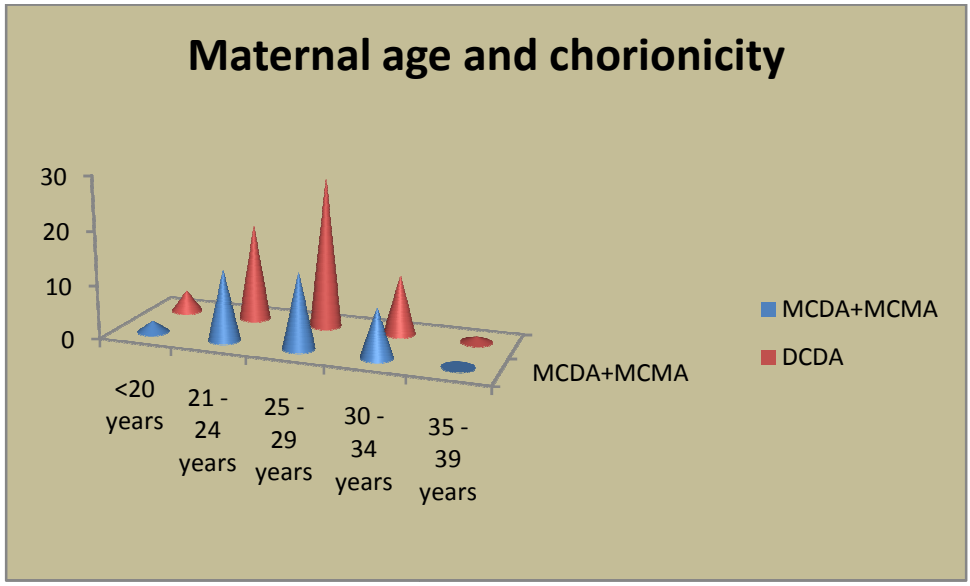


Out of the 100 twin pregnancies, primigravidas constituted for 43% of pregnancies and multigravidas constituted for 57%.

Table 2 showing distribution of 100 twin deliveries according to mother's age:

MATERNAL AGE IN YEARS	MCDA+MCMA	DCDA
<20 years	2	4
21 - 24 years	13	18
25 - 29 years	14	28
30 - 34 years	9	11
35 - 39 years	0	1

Figure 2 showing distribution of 100 twin deliveries according to mother's age:

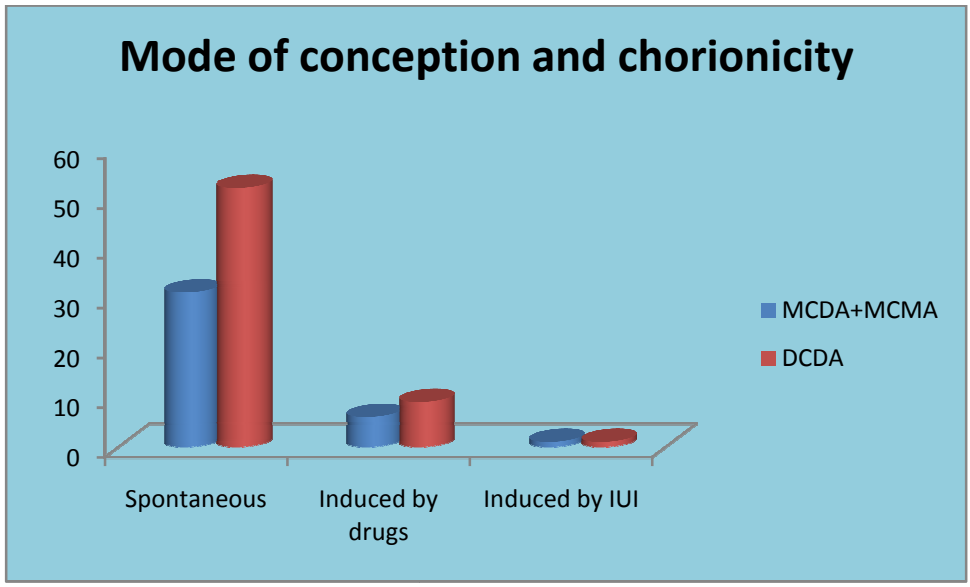


The most common age group for the incidence of twins according to our study was 25 to 29 years. In both mono and dichorionic pregnancies the common age group was 25 – 29 years. As the age increases, the incidence of dichorionicity is found to be increasing.

Table 3 showing distribution of 100 twin deliveries according to the mode of conception:

Mode of conception	MCDA+MCMA	DCDA
Spontaneous	31	52
Induced by drugs	6	9
Induced by IUI	1	1
Total	38	62

Figure 3 showing distribution of 100 twin deliveries according to the mode of conception:

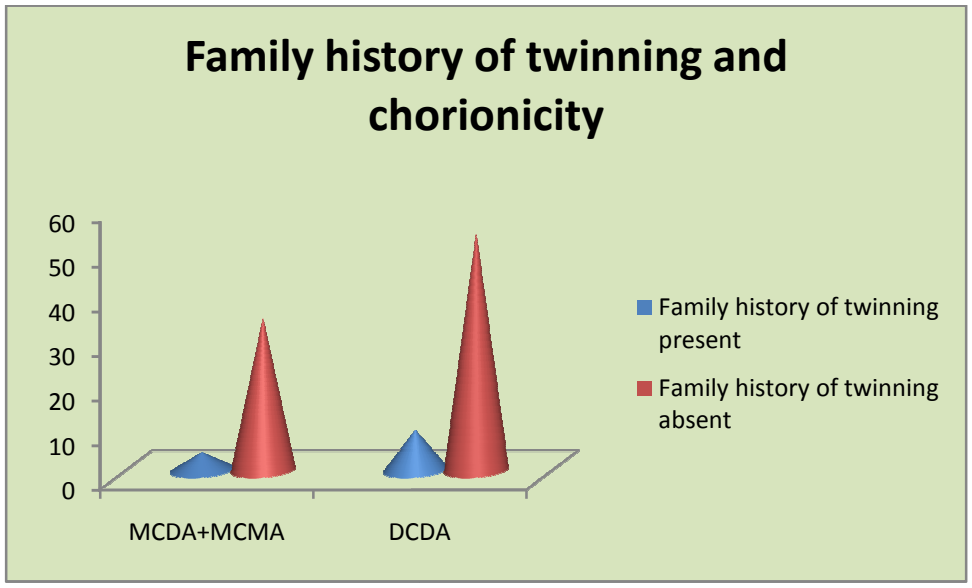


Out of the 100 twin pregnancies, 83% were out of spontaneous conception, 15% were induced by drugs and 2% were induced by IUI.

Table 4 showing family history of twinning in 100 twin deliveries:

Family history of twinning	MCDA+MCMA	DCDA
Family history of twinning present	4	9
Family history of twinning absent	34	53
Total	38	62

Figure 4 showing family history of twinning in 100 twin deliveries:

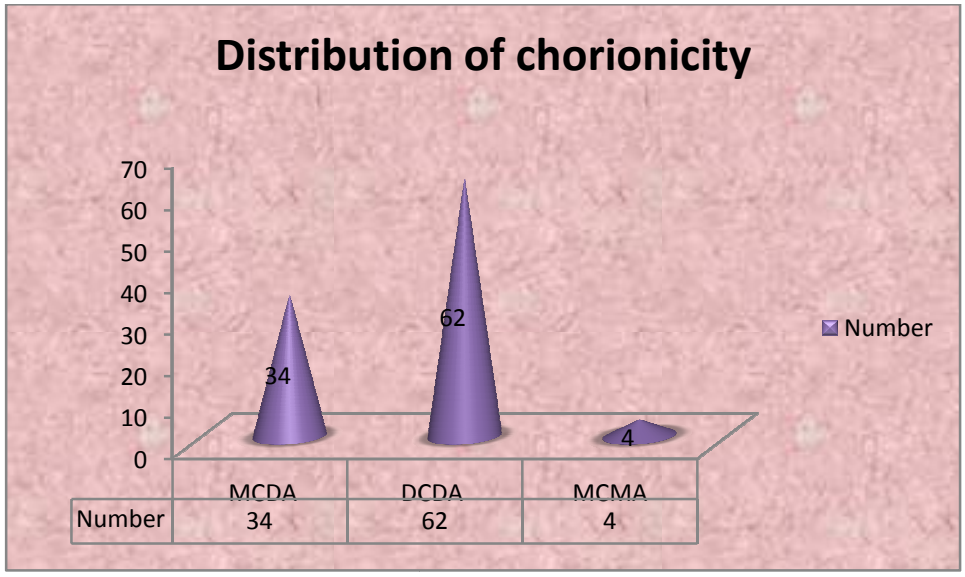


There was a positive family history of twinning in only 13% of twin pregnancies and it was absent in 87% of twins.

Table 5 showing the distribution of chorionicity among twin pregnancies:

Type of chorionicity	MCDA	DCDA	MCMA
Number	34	62	4
Percentage	34%	62%	4%

Figure 5 showing the distribution of chorionicity among twin pregnancies:

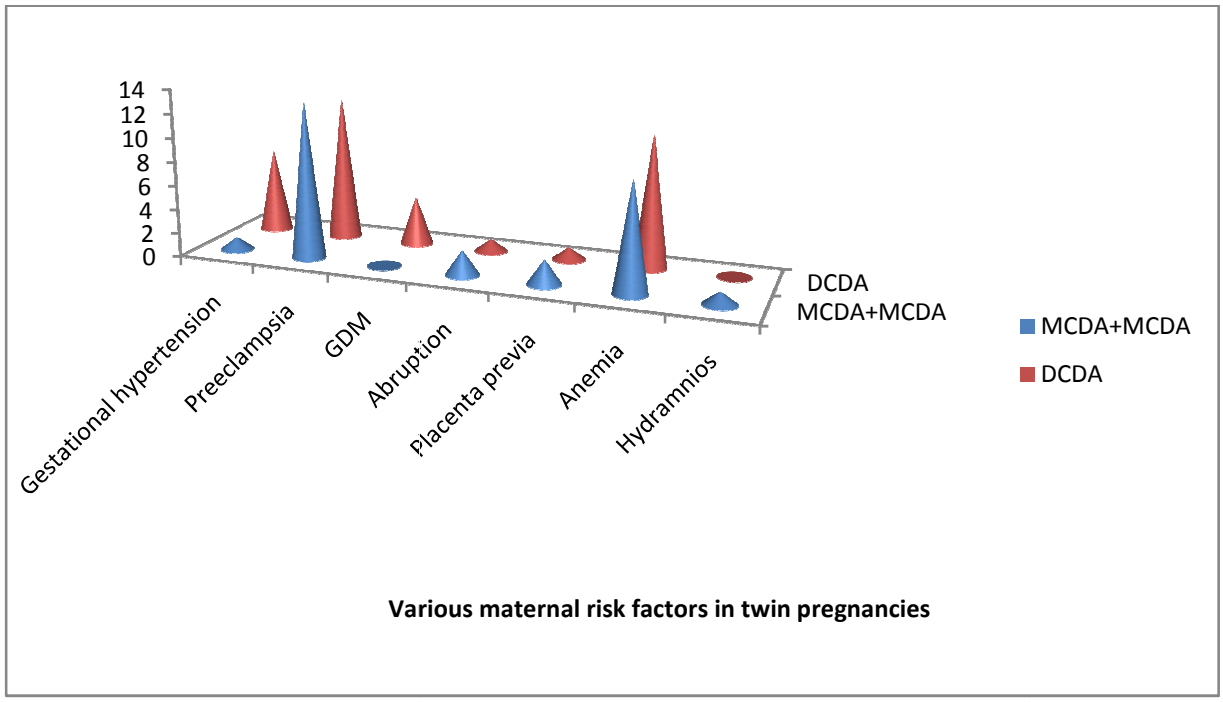


Among the 100 twin pregnancies, 62% were dichorionic diamniotic, 34% were monochorionic diamniotic and 4% were monochorionic and monoamniotic.

Table 6 showing various maternal risk factors complicating twin pregnancies:

Maternal risk factors	MCDA+MCDA	DCDA
Gestational hypertension	1	7
Preeclampsia	13	12
GDM	0	4
Abruption	2	1
Placenta previa	2	1
Anemia	9	11
Hydramnios	1	0

Figure 6 showing various maternal risk factors complicating twin pregnancies:



Gestational hypertension was present in 8%, preeclampsia was present in 25%, anemia was present in 20%, GDM was present in 4% and abruption, placenta previa in 3% and hydramnios in 1% of twin pregnancies.

Table 7 showing the preterm, preterm PPROM, PROM incidence in twin pregnancies:

Column1	MCDA+MCMA	DCDA
Term	1	34
Preterm	24	16
Preterm PPROM	12	5
PROM	1	7

Pre term complicating twin pregnancies was present in 44%, preterm PPROM in 17% and PROM in 8%. Out of the 38 monochorionic pregnancies, preterm was present in 63%, preterm PPROM was present in 31%, PROM was present in 2%. Out of the 62 dichorionic pregnancies, preterm was present in 25%, preterm PPROM was present in 8%, PROM was present in 11%. The significance of preterm between monochorionic and dichorionic pregnancies was analysed using the Fischer's exact test and it was found significant with a P value of <.05(0.0003). The significance of preterm PPROM between monochorionic and dichorionic pregnancies was analysed using the Fischer's exact test and it was found significant with a P value of <.05(0.0048). The significance of PROM between monochorionic and dichorionic pregnancies was analysed using the Fischer's exact test and it was not found significant with a P value of >.05.

Figure 7 showing the preterm, preterm PPROM, PROM incidence in twin pregnancies:

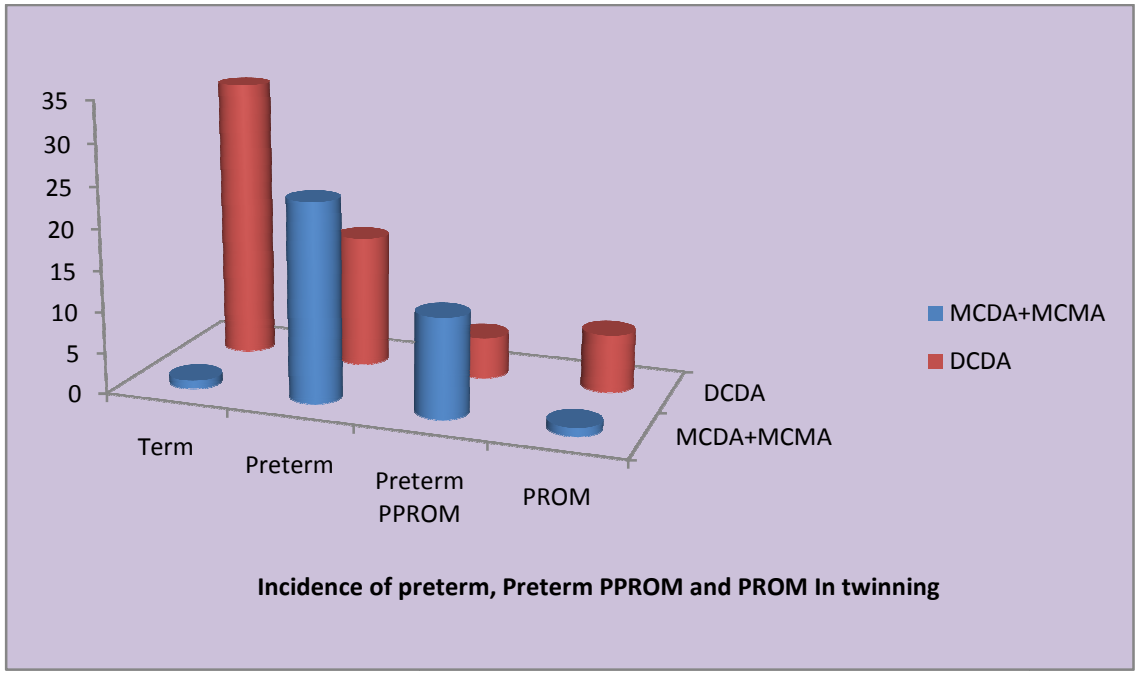


Table 8 showing gestational age of delivery according to chorionicity:

Gestational age in weeks	MCDA +MCMA	DCDA
28 - 30 weeks	10	1
31 - 33 weeks	18	2
34 - 36 weeks	9	25
>37 weeks	1	34

Out of the 100 twin deliveries, 34% took place around 34 – 36 weeks, 35% took place at a gestational age more than 37 weeks, 20% around 31- 33 weeks, and 11% in 28 – 30 weeks. Among the monochorionic pregnancies, 47% delivered at a gestational age of 31 -33 weeks, 23% delivered at 34 – 36 weeks, 2% delivered at more than 37 weeks and 26% delivered at 28 – 30 weeks. Among the dichorionic pregnancies, 3% delivered at a gestational age of 31 -33 weeks, 69% delivered at 34 – 36 weeks, 54% delivered at more than 37 weeks and 2% delivered at 28 – 30 weeks.

Figure 8 showing gestational age of delivery according to chorionicity:

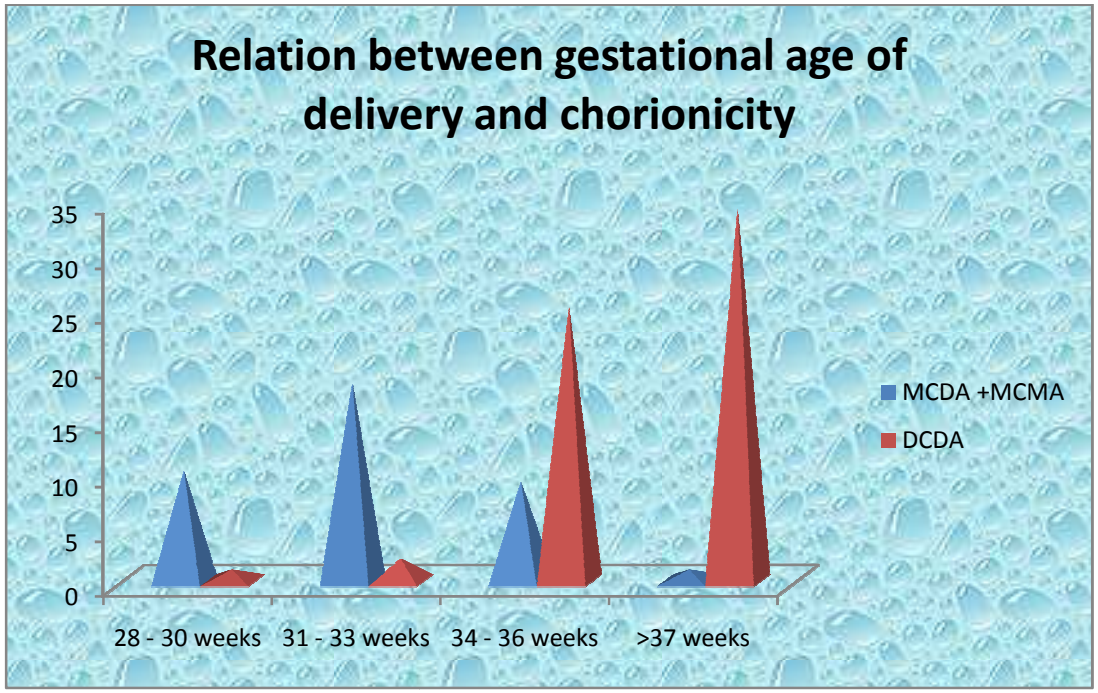


Table 9 showing the mode of delivery in 100 twin deliveries according to chorionicity:

MODE OF DELIVERY	MCDA+MCMA	DCDA
LSCS	25	37
Vaginal	10	22
Outlet vacuum	0	1
Vacuum extraction	1	0
Vaginal/LSCS	1	0
Forceps	1	2
Total	38	62

Out of the 38 monochorionic pregnancies, 65% was delivered by LSCS, 26% was delivered by vaginal, 3% was delivered by vacuum extraction, vaginal/LSCS, forceps each. Out of the 62 dichorionic pregnancies, 59% was delivered by LSCS, 35% was delivered by vaginal, 3% was delivered by vacuum extraction, outlet vacuum, vaginal/LSCS, forceps each.

Figure 9 showing the mode of delivery in 100 twin deliveries according to chorionicity:

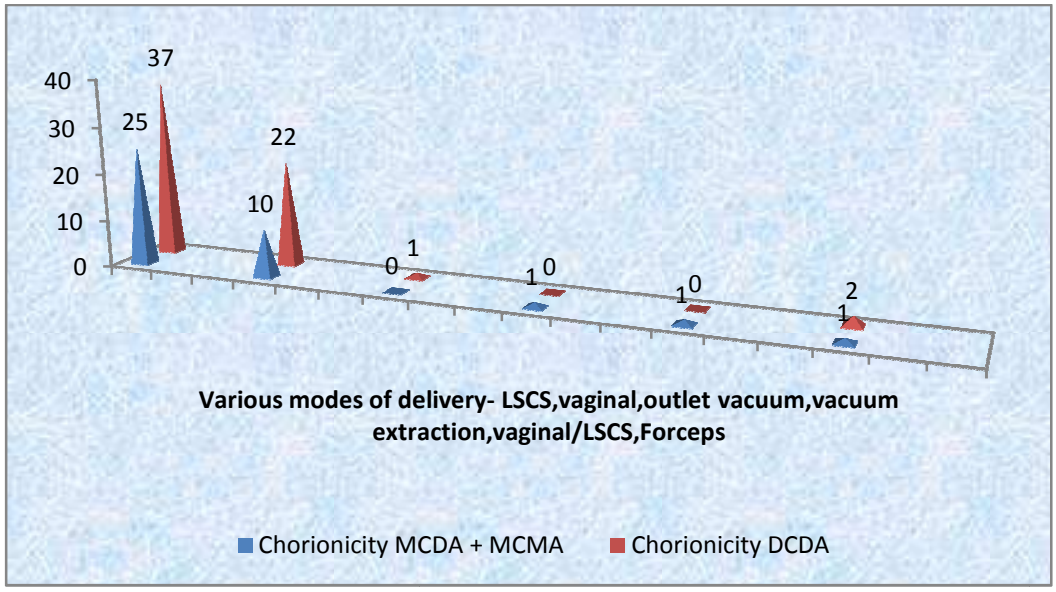
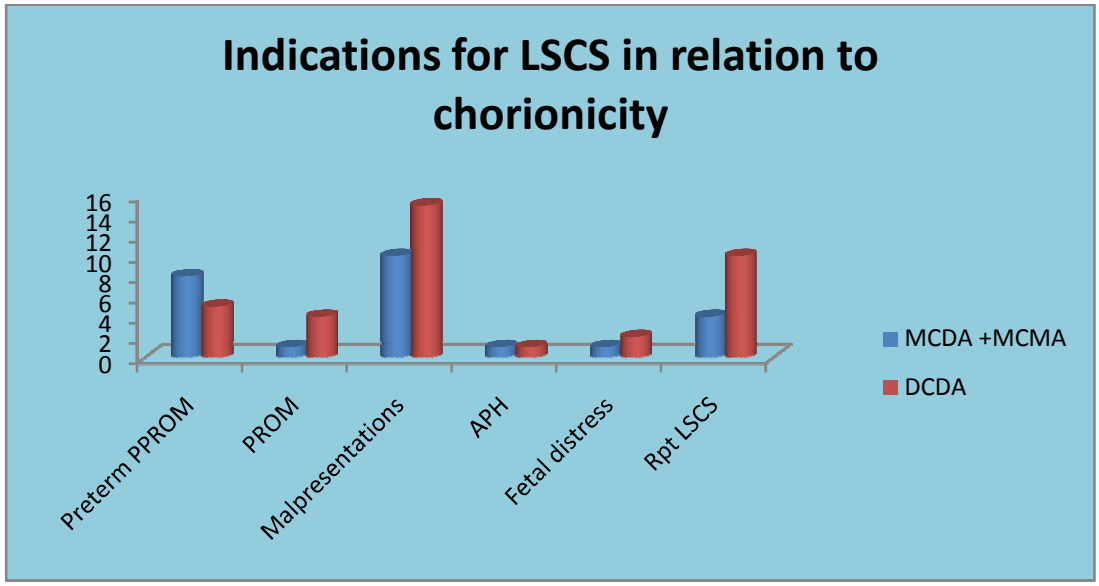


Table 10 showing various indications for LSCS in relation to chorionicity:

LSCS indications	MCDA +MCMA	DCDA
Preterm PPROM	8	5
PROM	1	4
Malpresentations	10	15
APH	1	1
Fetal distress	1	2
Rpt LSCS	4	10
Total	25	37

Figure 10 showing various indications for LSCS in relation to chorionicity:



The most common indication for LSCS was fetal malpresentations in both MC and DC pregnancies, followed by repeat LSCS and preterm PPROM.

Table 11 showing the various types of presentation in 100 twin deliveries:

Mode of presentation	MCDA+MCMA	DCDA
VxVx	14	26
VxBreech	9	13
BreechVx	8	11
VxTransverse	3	1
BreechTransverse	1	0
TransverseBreech	0	4
BreechBreech	3	7
Total	38	62

Out of the 100 twin deliveries, 40% was of both vertex presentation, 22% of Vx breech, 19% breech Vx, 8% of both breech, Vx transverse, transerve Breech was 4% each, 1% of breech transverse.

Figure 11 showing the various types of presentation in 100 twin deliveries according to chorionicity

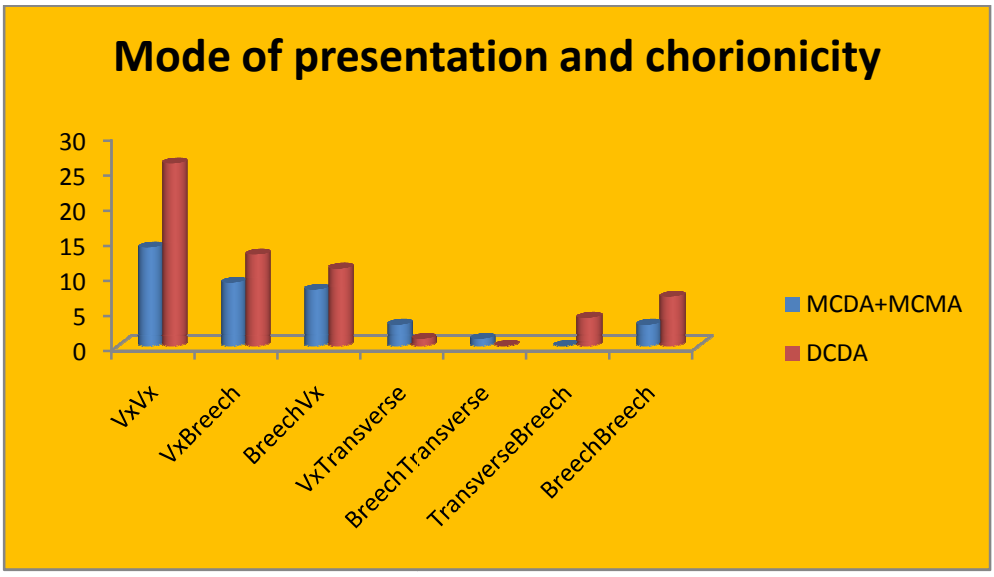


Table 12 showing relationship between PPH and chorionicity:

PPH	Chorionicity		Total
	MCDA + MCMA	DCDA	
Present	8	10	18
Absent	30	52	82
Total	38	62	100

Out of the 38 monochorionic pregnancies, PPH was present in 21% and out of 62 dichorionic pregnancies it was present in 16%. The significance of PPH with relation to chorionicity was compared was using Fischer's exact test and was not found to be significant with a P value of > 0.05.

Figure 12 showing relationship between PPH and chorionicity:

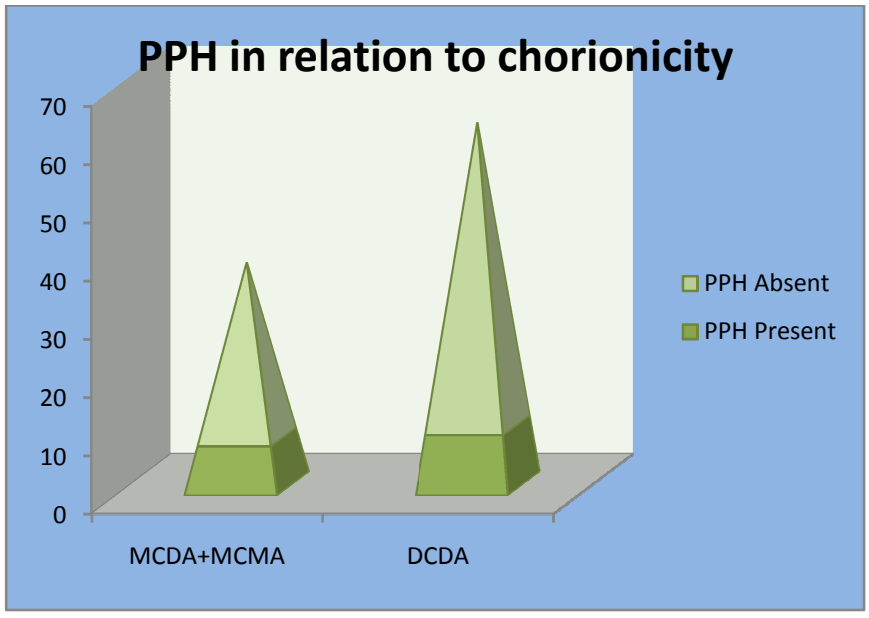


Table 13 showing relationship between intrauterine death and chorionicity:

IUD	Chorionicity		Total
	MCDA + MCMA	DCDA	
Present	7	3	10
Absent	69	121	190
Total	76	124	200

Out of 100 twin deliveries, IUD complicated 9% of monochorionicity and 2.4% of dichorionicity. The significance in difference between the two groups was analysed using the Fischer's test and was found significant with a P value of 0.0392.

Figure 13 showing relationship between intrauterine death and chorionicity:

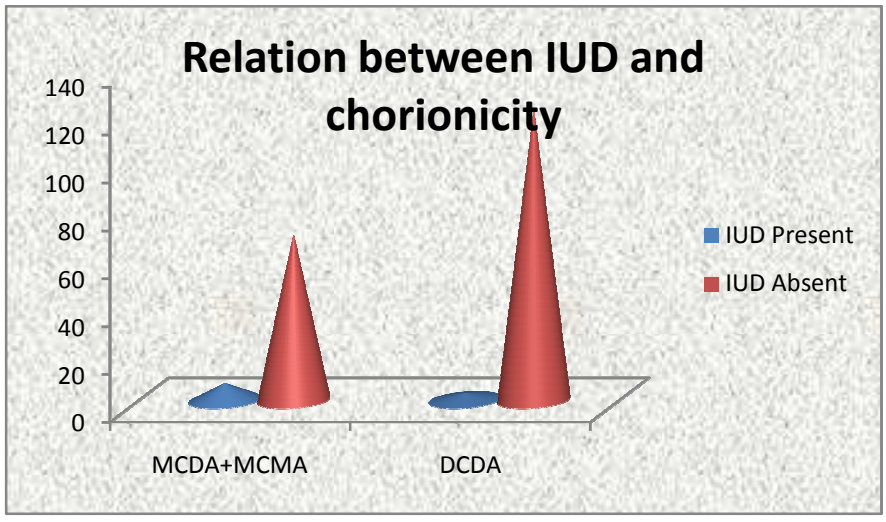


Table 14 showing the relationship between stillbirth and chorionicity:

Still birth	Chorionicity		Total
	MCDA + MCMA	DCDA	
Present	1	3	4
Absent	75	121	196
Total	76	124	200

Out of 100 twin deliveries, still birth was found in 1% of 76 monochorionic pregnancies and 2% of dichorionic pregnancies. Using the fischer's exact test no significant difference was observed in the rates of still birth between MC and DC pregnancies.

Figure 14 showing the relationship between stillbirth and chorionicity:

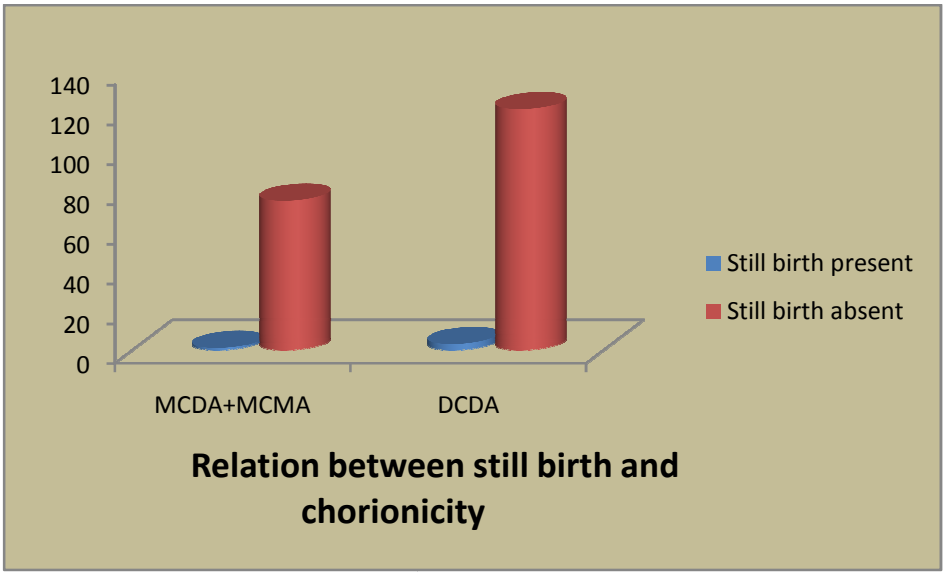


Table 15 showing the relation between birth weight and chorionicity:

Birth Weight	Chorionicity		Total
	MCDA + MCMA	DCDA	
< 1.5	25	13	38
1.5-2.5	40	80	120
> 2.5	11	31	42
Total	76	124	200

Out of 100 twin deliveries, 32% in monochorionic pregnancies and 10% in dichorionic pregnancies were found to have a birth weight of less than 1.5 kg, 52% in MC and 64% in DC had a birth weight between 1.5 – 2.5 kgs, 14% in MC and 25% in DC had a birth weight more than 2.5 kgs.

Figure 15 showing the relation between birthweight and chorionicity:

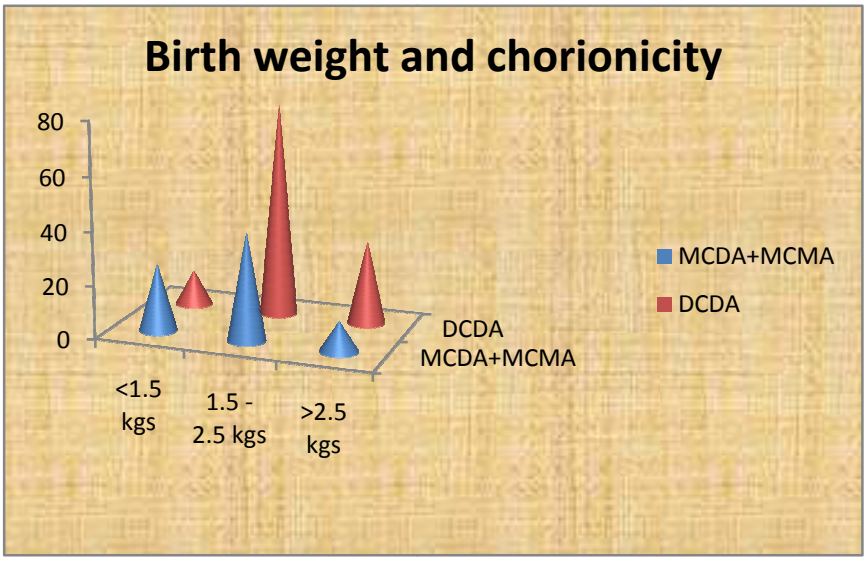


Table 16 showing the relation between congenital anomalies and chorionicity:

Congenital Anomalies	Chorionicity		Total
	MCDA + MCMA	DCDA	
Present	9	1	10
Absent	67	123	190
Total	76	124	200

Out of the monochorionic pregnancies, congenital anomalies was present in 12% and in 0.8% of DC pregnancies. The P value was found to be 0.0008.

Figure 16 showing the relation between congenital anomalies and chorionicity:

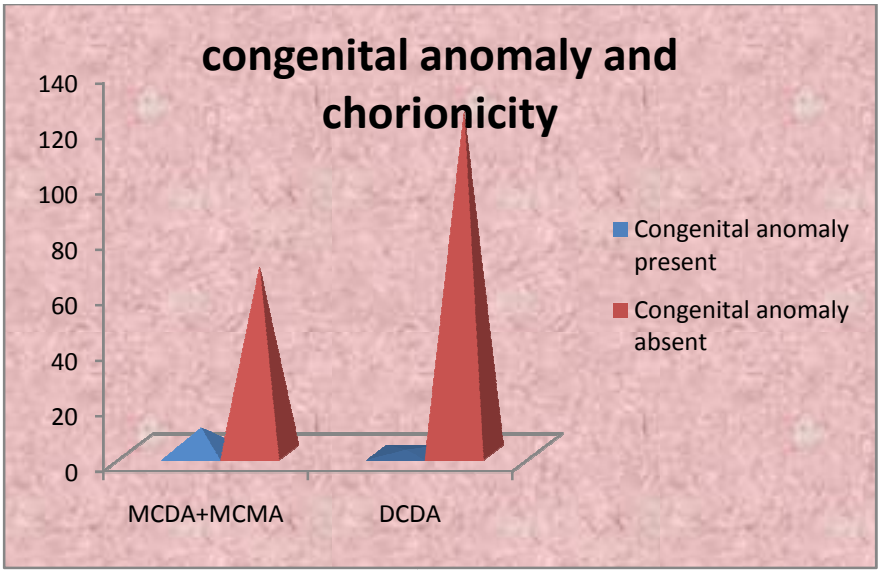


Table 17 showing relation between IUGR and chorionicity:

Selective IUGR	Chorionicity		Total
	MCDA + MCMA	DCDA	
Present	7	2	9
Absent	69	122	191
Total	76	124	200

Out of the monochorionic pregnancies, IUGR was present in 9.2% and in 1.6% of DC pregnancies. The P value was found to be 0.0008.

Figure 17 showing relation between IUGR and chorionicity:

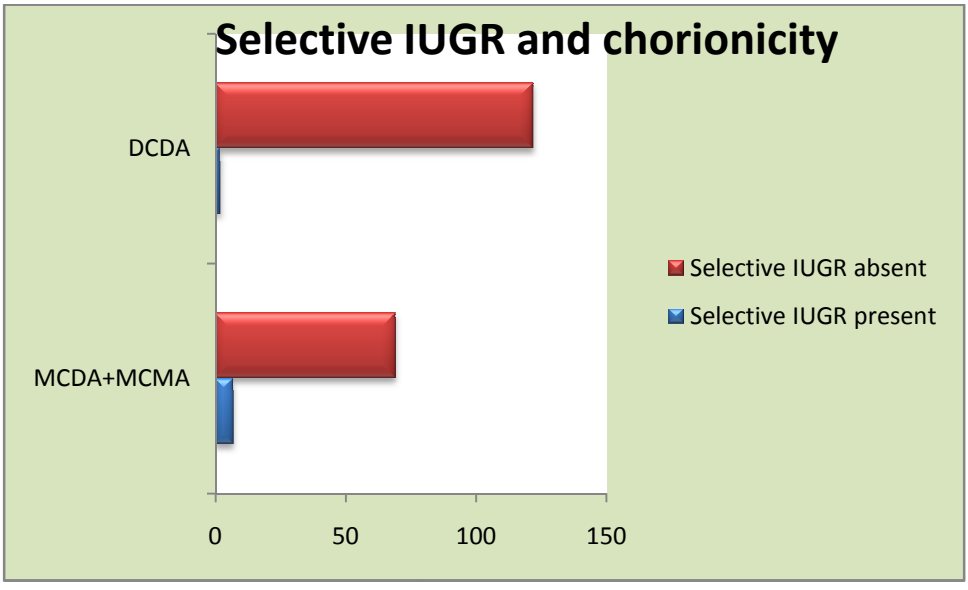


Table 18 showing relation between apgar score and chorionicity:

5 minute Apgar	Chorionicity		Total
	MCDA + MCMA	DCDA	
0-5	15	17	32
6-7	35	28	63
8-10	26	79	115
Total	76	124	200

Out of MC pregnancies, a 5 minute apgar score of less than 7 was found in 65% and 37% of DC pregnancies. The P value between the two groups was found significant at 0.0001.

Figure 18 showing relation between apgar score and chorionicity:

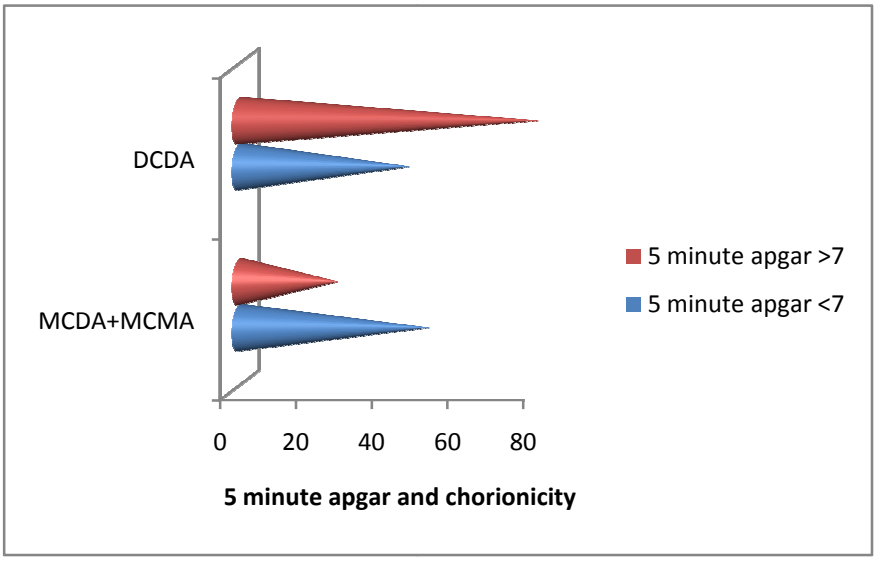


Table 19 showing relation between discordant growth and chorionicity:

Discordant growth	Chorionicity		Total
	MCDA + MCMA	DCDA	
Present	15	7	22
Absent	61	117	178
Total	76	124	200

Out of MC pregnancies, discordant growth was found in 19% and 5% of DC pregnancies. The P value between the two groups was found significant at 0.0042.

Figure 19 showing relation between discordant growth and chorionicity:

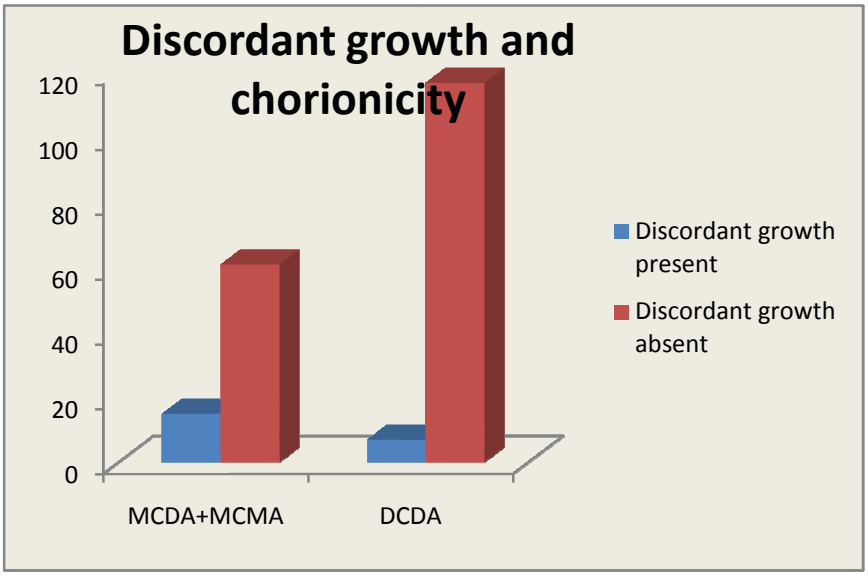


Table 20 showing relation between neonatal morbidity and chorionicity

CAUSES OF NEONATAL MORBIDITY	MCDA+MCMA	DCDA
RDS	27	18
LBW	20	11
Birth asphyxia	3	6
VLBW	10	8
Hyperbilirubinemia	1	4
CHD	1	0
Hypoglycemia	0	1
Total	62	48

Causes for neonatal morbidity like RDS was present in 27% of MC and 13% of DC, LBW was present in 26& of MC and 8% of DC, VLBW in 13% of MC and 6.4% of DC, birth asphyxia in 3% of MC and 4.8% of DC, hyperbilirubinemia in 1.3% of MC and 3.2% of DC and CHD in 1.3% of MC and 0.8% of DC, hypoglycaemia in 0% of MC and 0.8% of DC pregnancies.

Figure 20 showing relation between neonatal morbidity and chorionicity:

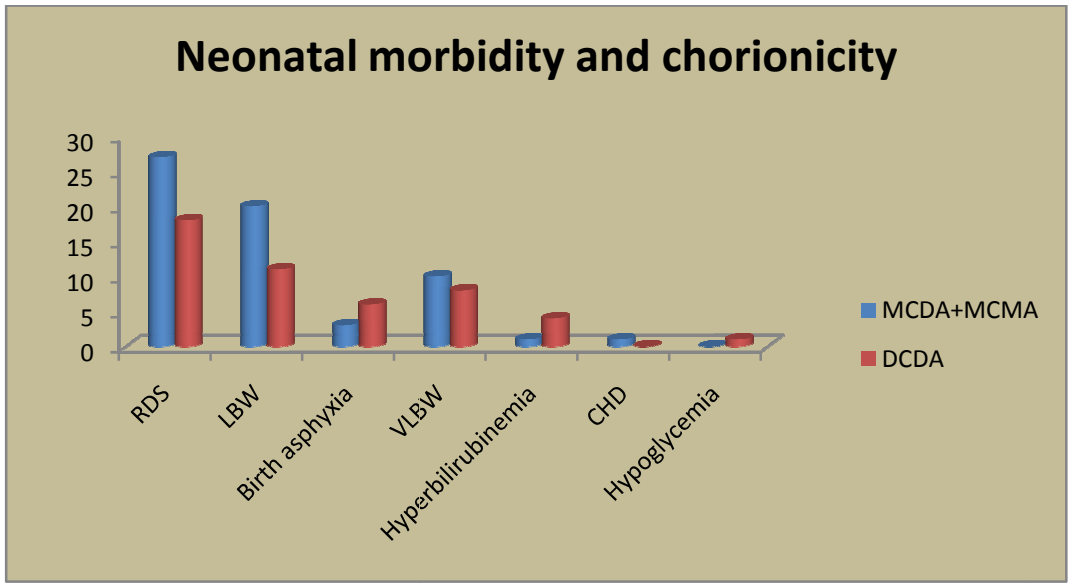


Table 21 showing relation between neonatal mortality and chorionicity:

Neonatal Death	Chorionicity		Total
	MCDA + MCMA	DCDA	
Present	15	9	24
Absent	61	115	176
Total	76	124	200

Out of MC pregnancies, neonatal mortality was found in 19% and 7.2% of DC pregnancies. The P value between the two groups was found significant at 0.0126.

Figure 21 showing relation between neonatal mortality and chorionicity:

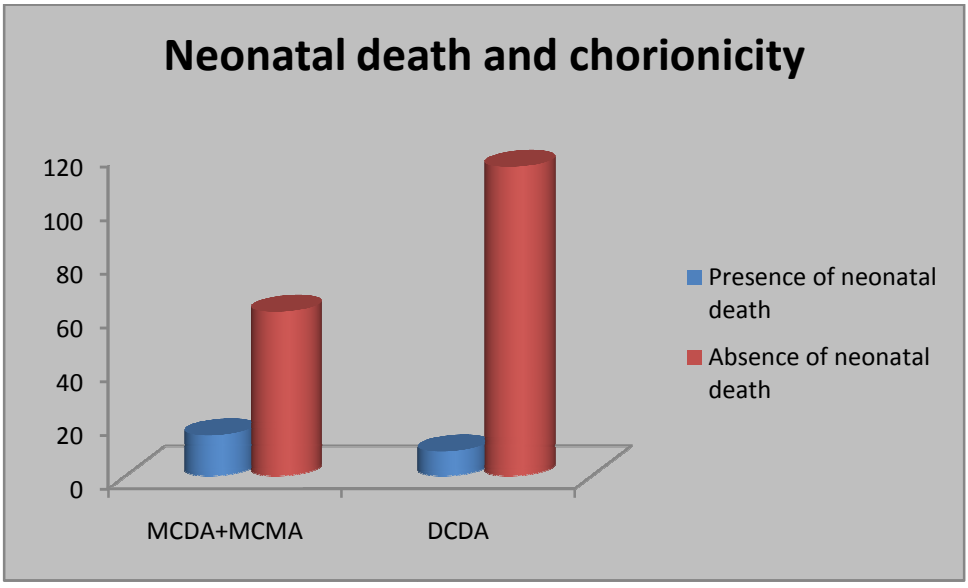


Table 22 showing relation between causes of neonatal death and chorionicity:

CAUSE OF DEATH	MCDA+MCMA	DCDA
RDS& sepsis	7	4
Sepsis	3	1
Birth asphyxia	1	1
seizures	1	1
NEC	1	1
IVH	1	1
CHD	1	0
Total	15	9

Out of 15 neonatal deaths in MC & 9 deaths in DC, RDS & sepsis constituted for 46% in MC and 44% in DC, sepsis for 26% in MC and 11% in DC, birth asphyxia, seizures, NEC, IVH for 6% in MC and DC. CHD accounted for death in 6% of MC.

Figure 22 showing relation between causes of neonatal death and chorionicity:

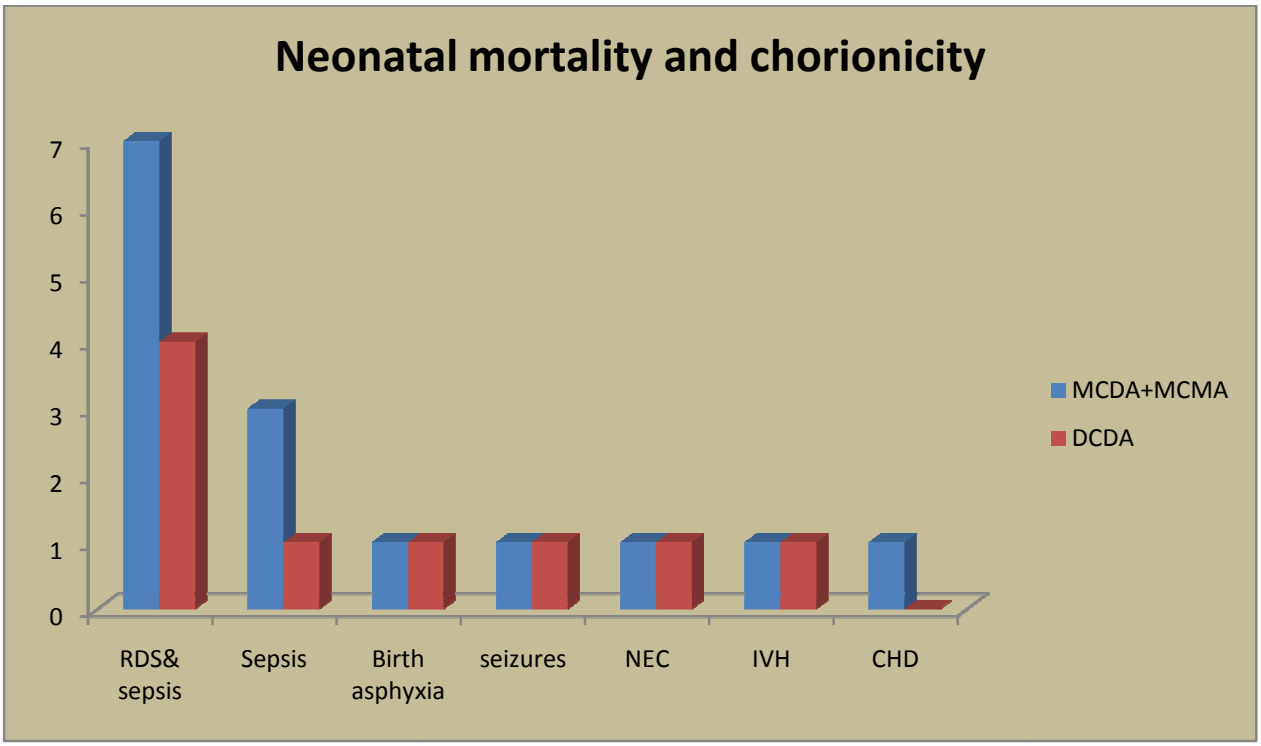


Table 23 showing relation between no. of days of NICU admission and chorionicity:

No.of days of NICU admission	MCDA+MCMA	DCDA	Total
1-5 days	10	24	34
6-10 days	15	10	25
11-15 days	10	6	16
16- 20 days	8	2	10
21-30 days	5	0	5
Total	48	42	90

Figure 23 showing relation between no. of days of NICU admission and chorionicity:

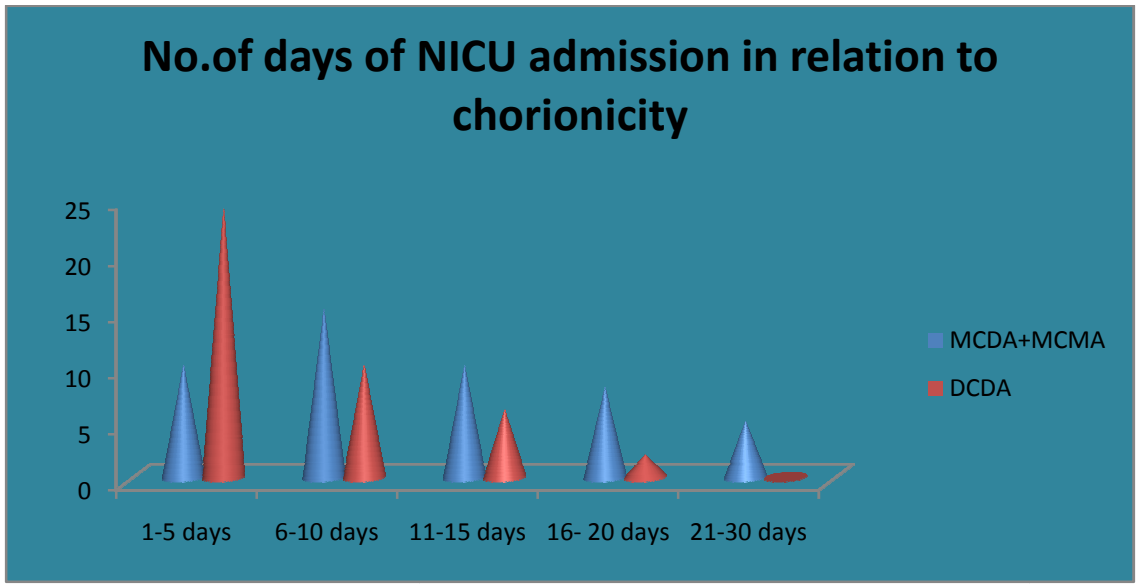
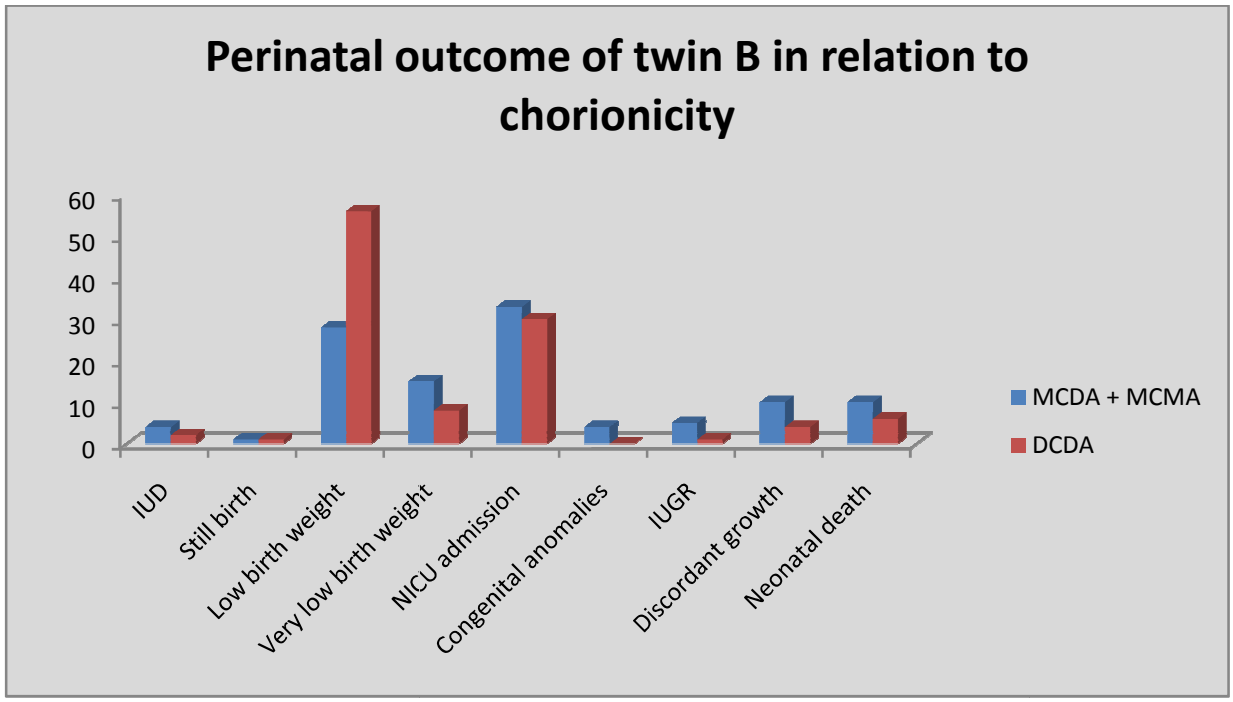


Table 24 showing the perinatal outcome of twin B in relation to chorionicity:

Perinatal outcome of twin B	MCDA + MCMA	DCDA	P value
IUD	4	2	0.1971
Still birth	1	1	1
Low birth weight	28	56	0.0467
Very low birth weight	15	8	0.0032
NICU admission	33	30	0.0001
Congenital anomalies	4	0	0.0188
IUGR	5	1	0.0284
Discordant growth	10	4	0.0078
Neonatal death	10	6	0.047

Figure 24 showing the perinatal outcome of twin B in relation to chorionicity:



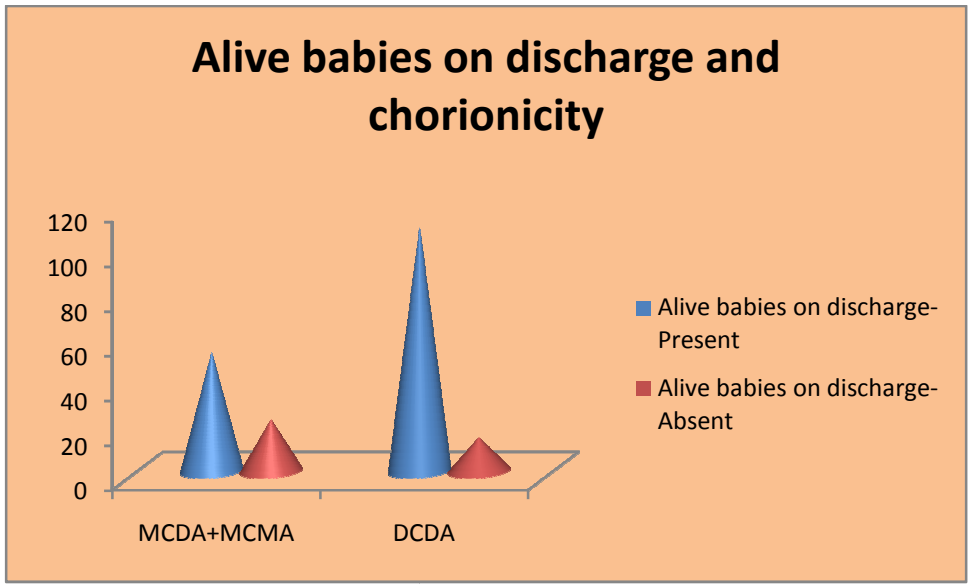
There was no significant difference between MC and DC twin B as far as IUD and still birth was considered. However LBW, VLBW, NICU admission, congenital anomalies, IUGR, discordant growth and neonatal death were significantly higher in MC twin B than DC twin B.

Table 25 showing relation between no. of live babies on discharge and chorionicity:

Alive babies on discharge	MCDA+MCMA	DCDA	Total
Present	53	109	162
Absent	23	15	38
Total	76	124	200

Out of 38 MC pregnancies, 69% of babies were discharged alive and in 62 DC pregnancies, 87% of babies were discharged alive. The P value between the two groups was significant with a value of 0.0026.

Figure 25 showing relation between no. of live babies on discharge and chorionicity:



DISCUSSION

We conducted a study on 100 twin pregnancies admitted in ISO KGH. The aim of the study was to determine the perinatal outcome according to chorionicity.

Among the 100 twin pregnancies, 62% were dichorionic diamniotic, 34% were monochorionic diamniotic and 4% were monochorionic and monoamniotic. Studies by Assuncao et al[45] conducted in 289 twin pregnancies between 2003 to 2006 it was found that 60% were DCDA, 30.8% were MCDA and 6.6% were MCMA. Our study results were similar to this study.

Out of the 100 twin pregnancies, primigravidas constituted for 43% of pregnancies and multigravidas constituted for 57%. As the parity increases, the incidence of dichorionicity is found to increase. The result was similar to the study conducted by Azubike et al in Nigeria during the year 1982[46] which showed that as parity advances, the incidence of twin increases from 2% in primi to 6.6% in multiparous women.

The most common age group for the incidence of twins according to our study was 25 to 29 years. In both mono and dichorionic pregnancies the common age group was 25 – 29 years. As the age increases, the incidence of dichorionicity is

found to be increasing. Study by Summera alsam et al[47] conducted at Lahore had a similar incidence of twinning at the age group of around 25 years.

Out of the 100 twin pregnancies, 83% were out of spontaneous conception, 15% were induced by drugs and 2% were induced by IUI. There was no difference with regard to mode of conception between MC and DC pregnancies. This result was similar to the Study by Assuncao et al[45].

There was a positive family history of twinning in only 13% of twin pregnancies and it was absent in 87% of twins.

When maternal complications were analysed, Gestational hypertension was present in 8%, preeclampsia was present in 25%, anemia was present in 20%, GDM was present in 4% and abruption, placenta previa in 3% and hydramnios in 1% of twin pregnancies. The corresponding figures reported by Chaudhary et al[48] were 22.6% for hypertension, 35.8% for anemia, 5.7% for APH and 5.7% for polyhydramnios. There was no difference in the presence of maternal risk factors among MC and DC pregnancies.

Other maternal complications like preterm, preterm PPROM and PROM were analysed separately. Out of the 38 monochorionic pregnancies, preterm was present in 63%, preterm PPROM was present in 31%, PROM was present in 2%.

Out of the 62 dichorionic pregnancies, preterm was present in 25%, preterm PPROM was present in 8%, PROM was present in 11%. The significance of difference in preterm incidence between monochorionic and dichorionic pregnancies was analysed using the Fischer's exact test and it was found significant with a P value of <.05(0.0003). This was contrast to the study by Summera et al[47] where preterm incidence in MC and DC pregnancies were similar. The significance of difference in preterm PPROM incidence between monochorionic and dichorionic pregnancies was analysed using the Fischer's exact test and it was found significant with a P value of <.05(0.0048). The significance of difference in incidence of PROM between monochorionic and dichorionic pregnancies was analysed using the Fischer's exact test and it was not found significant with a P value of >.05.

Gestational age at delivery was analysed. The mean gestational age in our study was 34.4 weeks similar to study by Assuncao et al[45]. Among the monochorionic pregnancies, 47% delivered at a gestational age of 31 -33 weeks, 23% delivered at 34 – 36 weeks, 2% delivered at more than 37 weeks and 26% delivered at 28 – 30 weeks. Among the dichorionic pregnancies, 3% delivered at a gestational age of 31 -33 weeks, 69% delivered at 34 – 36 weeks, 54% delivered at more than 37 weeks and 2% delivered at 28 – 30 weeks. Hence it was inferred that the mean

gestational age at delivery was lower in MC pregnancies compared to DC similar to the study by Assuncao et al[45].

Next the mode of delivery was analysed. Out of the 38 monochorionic pregnancies, 65% was delivered by LSCS, 26% was delivered by vaginal, 3% was delivered by vacuum extraction, vaginal/LSCS, forceps each. Out of the 62 dichorionic pregnancies, 59% was delivered by LSCS, 35% was delivered by vaginal, 3% was delivered by vacuum extraction, outlet vacuum, vaginal/LSCS, forceps each. This was similar to the study by Assuncao et al[45] where the incidence of LSCS were more than vaginal deliveries. Studies by Khali et al, Summera Alsam et al also had similar results.

The mode of presentation was analysed. Out of the 100 twin deliveries, 40% was of both vertex presentation, 22% of Vx breech, 19% breech Vx, 8% of both breech, Vx transverse, transerve Breech was 4% each, 1% of breech transverse. The most common mode of presentation was vertex/vertex in both MC and DC pregnancies which was similar to the results in the study conducted by PA Hatkar et al[49].

PPH as a complication was analysed separately. Out of the 38 monochorionic pregnancies, PPH was present in 21% and out of 62 dichorionic pregnancies it was present in 16%. The significance of PPH with relation to chorionicity was

compared was using Fischer's exact test and was not found to be significant with a P value of > 0.05. This was similar to the results in study by Naushaba et al[50].

IUD was assessed. Out of 100 twin deliveries, IUD complicated 9% of monochorionicity and 2.4% of dichorionicity. The significance in difference between the two groups was analysed using the Fischer's test and was found significant with a P value of 0.0392. The results were similar to Assuncao et al[45], summera et al[47] where IUD in MC pregnancies were more in DC pregnancies.

Out of 100 twin deliveries, still birth was found in 1% of 76 monochorionic pregnancies and 2% of dichorionic pregnancies. Using the fischer's exact test no significant difference was observed in the rates of still birth between MC and DC pregnancies. This is in contrast to the results of the study by Svetlana et al[51] who showed increased still birth among MC pregnancies.

Out of 100 twin deliveries, 32% in monochorionic pregnancies and 10% in dichorionic pregnancies were found to have a birth weight of less than 1.5 kg, 52% in MC and 64% in DC had a birth weight between 1.5 – 2.5 kgs, 14% in MC and 25% in DC had a birth weight more than 2.5 kgs. MC twins had a lower birth weight compared to DC twins similar to the study results by PA Hatkar et al[49].

Babies with birth weight less than 1.5 kgs were found to occur at a higher rate in MC than in DC pregnancies.

Out of the monochorionic pregnancies, congenital anomalies were present in 12% and in 0.8% of DC pregnancies. The P value was found to be 0.0008. The incidence of congenital anomalies were more in MC pregnancies similar to the results shown by S.V.Glinianaia et al who showed that congenital anomalies were twice as common in MC pregnancies as in DC pregnancies[52].

Out of the monochorionic pregnancies, IUGR was present in 9.2% and in 1.6% of DC pregnancies. The P value was found to be 0.0008. The incidence of IUGR was found to be statistically significant in MC pregnancies similar to the study results of Dominigues et al[53].

Out of MC pregnancies, a 5 minute apgar score of less than 7 was found in 65% and 37% of DC pregnancies. The P value between the two groups was found significant at 0.0001. The apgar score was found to be less in MC pregnancies similar to the study results of Naushaba et al[50].

Out of MC pregnancies, discordant growth was found in 19% and 5% of DC pregnancies. The P value between the two groups was found significant at 0.0042.

Discordancy was more in MC pregnancies similar to the results of Domonigues et al[53].

Causes for neonatal morbidity like RDS was present in 27% of MC and 13% of DC, LBW was present in 26& of MC and 8% of DC, VLBW in 13% of MC and 6.4% of DC, birth asphyxia in 3% of MC and 4.8% of DC, hyperbilirubinemia in 1.3% of MC and 3.2% of DC and CHD in 1.3% of MC and 0.8% of DC, hypoglycaemia in 0% of MC and 0.8% of DC pregnancies. The most common cause for morbidity was determined as RDS due to prematurity. All the complications were found to occur at a higher rate in MC pregnancies as in the study by Dominigues et al[53].

Out of MC pregnancies, neonatal mortality was found in 19% and 7.2% of DC pregnancies. The P value between the two groups was found significant at 0.0126. This was comparable to the study results of Summera Alsam et al[47].

Out of 15 neonatal deaths in MC & 9 deaths in DC, RDS & sepsis constituted for 46% in MC and 44% in DC, sepsis for 26% in MC and 11% in DC, birth asphyxia, seizures, NEC, IVH for 6% in MC and DC. CHD accounted for death in 6% of MC. The most common cause for death among both groups was RDS & sepsis secondary to prematurity.

The number of days in NICU was analysed and it was found that MC deliveries had a longer duration of stay (mean = 18.5 days) compared to DC where the mean duration of stay was 5.6 days.

Out of 38 MC pregnancies, 69% of babies were discharged alive and in 62 DC pregnancies, 87% of babies were discharged alive. The P value between the two groups was significant with a value of 0.0026. Monochorionic pregnancies had a lesser probability of getting discharged alive compared to DC pregnancies.

SUMMARY

100 twin pregnancies were studied in our institute. Out of 100 twin pregnancies, 62% were DCDA, 34% were MCDA and 4% were MCMA.

The incidence of twin pregnancies in multigravida was 57% and in primi it was 43% and the incidence of dichorionicity increased as parity increased.

In both MCDA and DCDA, the common age group made out in our study was 25 to 29 years. The dichorionicity was found to increase as age advanced.

There was no difference in the mode of conception and family history with regard to chorionicity.

Of the maternal antepartum complications, preeclampsia ranked first which was present in about 25%, anemia occurred in 20%, GDM in 4% and APH in 3% and hydramnios in 1% out of the 100 twin deliveries. There was no statistical difference in maternal complications between MCDA and DCDA.

Preterm birth constituted for about 63% and preterm pprom in 31% of monochorionic twins and there was statistical significance between MCDA and DCDA. It constituted the major cause for neonatal morbidity and mortality.

The mean gestational age of delivery in our study for MCDA was 33 weeks compared to DCDA which was 34 weeks. The mean gestational age for MCDA was lower than DCDA.

LSCS was the most common mode of delivery for both MCDA and DCDA followed by vaginal delivery in our study.

Among the postpartum complications, PPH was present in 21% of MCDA

and 16% of DCDA which was statistically significant.

Of the specific complication in relation to chorionicity, the incidence of IUD, selective IUGR , congenital anomalies ,discordant growth were more in MCDA and there was statistical difference between the two groups. There was no statistical difference in still births between the two groups and this shows there are other reasons for stiibirth in dichorionicity.

The incidence of very low birth was more in MCDA (32%) compared to DCDA (10%) and it results in increased mortality.

The fetal outcome in terms of NICU admission, number of twins expired, apgar score <7, number of babies discharged alive showed statistical

significance between MCDA and DCDA and more adverse outcome was noted in MCDA.

The most common cause for neonatal death for both MCDA and DCDA in our study was RDS and sepsis secondary to prematurity, followed by very low birth weight.

CONCLUSION

- The incidence of maternal and fetal adverse outcome are increased significantly in twin pregnancies.
- MC pregnancies are at increased risk of developing various complications.
- It is highly advisable to determine the chorionicity at 11-14 weeks of gestation as each type of placentation carries different prognosis and morbidity.
- By determining the placentation we can counsel the parents regarding the risk of adverse perinatal outcome and invasive testing.
- Assessment of chorionicity helps in the management of discordant growth, twin to twin transfusion, feasibility of multifetal reduction and management of other complications.
- Early diagnosis of chorionicity and proper follow up throughout the gestation improves the perinatal outcome.
- Regular ultrasound study and if needed Doppler study for the growth and wellbeing of the twins particularly monochorionic twins is mandatory.
- Early detection and management of preterm, early referral to fetal medical centers in case if complication occurs and early hospitalization

are the most important steps in improving perinatal outcome and reducing adverse maternal outcome in cases of twin pregnancies.

BIBLIOGRAPHY

1. Assuncao RA, Liao AW, Brizot ML, et al., Perinatal outcome of twin pregnancies delivered in a teaching hospital. Rev Assoc Med Bras 2010; 56(4): 447-451.
2. Jacques Milliez, M.D. et al. Ethical aspects of multiple Pregnancy. International Journal of Fertility and Sterility. Vol.3, No.1, May-Jun 2009, Pages: 41-46.
3. Chitrit Y, Filidori M, Pons JC, Duyme M and Papiernik E. Perinatal mortality in twin pregnancies: a 3-year analysis in Seine Saint-Denis (France). Eur J Obstet Gynecol Reprod Med 1999;86:23-8.
4. Snijder MJ and Wladimiroff JW. Fetal biometry and outcome in monochorionic vs. dichorionic twin pregnancies; a retrospective cross-sectional matched-control study. Ultrasound Med Biol 1998;24:197-201.
5. Sebire NJ, Snijders RJ, Hughes K, Sepulveda W and Nicolaides KH. The hidden mortality of monochorionic twin pregnancies. Br J Obstet Gynaecol 1997;104:1203-7.
6. Victoria A, Mora G and Arias F. Perinatal outcome, placental pathology, and severity of discordance in monochorionic and dichorionic twins. Obstet Gynecol 2001;97:310-5.
7. Ong SS, Zamora J, Khan KS and Kilby MD. Prognosis for the co-twin following single-twin death: a systematic review. BJOG 2006;113:992-8.
8. Pharoah PO and Adi Y. Consequences of in-utero death in a twin pregnancy. Lancet 2000;355:1597-602.
9. Anna dera et al, Twin pregnancy – physiology, complications and mode of delivery, Archives of perinatal medicine; 13,(3), 7 – 16, 2007.
10. Sandler T.W. (1995) Langman's Medical Embryology; Fetal Membranes and Placenta: 101-21.

11. Errol R. Norwitz, Maternal Physiology and Complications of Multiple Pregnancy; Semin Perinatol 29:338-348.

12. Pritchard JA: Changes in the blood volume during pregnancy and delivery. Anesthesiology 26:393-399, 1965

13. Backus Chang A. (2004) Physiologic Changes of Pregnancy W: Obstetric Anesthesia Principles and Practice Red. Chestnut D.H. EM: 15-37.

14. Davison JM, Vallotton MB, Lindheimer MD: Plasma osmolality and urinary concentration and dilution during and after pregnancy. Br J Obstet Gynaecol 88:472-479, 1981

15. Hellgren M. (1996) Hemostasis during pregnancy and puerperium Hemostasis 26: 244-7.

16. Cunningham, Leveno et al., Williams obstetrics, 23 rd edition, chapter 39; 859 – 85.

17. Johanne Dube et al. Does chorionicity or zygosity predict adverse perinatal outcomes in twins?Am J Obstet Gynecol 2002;186:579-83.

18. Karien Hack, Monochorionic twin pregnancies. Thesis, University Utrecht, The Netherlands, Chapter 2.

19. Ana Patrícia Domingues et al, Chorionicity in twin pregnancies: impact upon perinatal results: Acta Obstet Ginecol Port 2007;1(4):163-166.

20. Dias T et al, Cord entanglement and perinatal outcome in monoamniotic twin pregnancies, Ultrasound Obstet Gynecol. 2010 Feb;35(2):2014.

21. Nihal Al Riyami et al, Perinatal Outcome of Monochorionic in Comparison to Dichorionic Twin Pregnancies, Oman Med J. 2013 May; 28(3): 173–177.

22. Victoria A et al, Perinatal outcome, placental pathology, and severity of discordance in monochorionic and dichorionic twins, Obstet Gynecol. 2001 Feb;97(2):310-5.

23. Sivan E et al. Impact of fetal reduction on the incidence of gestational diabetes. Obstet Gynecol 2002;99:91–4.

24. Roach VJ et al.. The incidence of gestational diabetes in multiple pregnancy. Aust N Z J Obstet Gynaecol 1998;38:56–7.

25. Sibai BM et al. Hypertensive disorders in twin versus singleton gestations. National Institute of Child Health and Human Development Network of Maternal-Fetal Medicine Units. Am J Obstet Gynecol 2000;182:938–42.

26. Mastrobattista JM et al. The rate of severe preeclampsia is increased in triplet as compared to twin gestations. Am J Perinatol 1997;14:263–5.

27. Krotz S et al. Hypertensive disease in twin pregnancies: a review. Twin Res 2002;5:8–14.

28. Long PA, Oats JN. Preeclampsia in twin pregnancy—severity and pathogenesis. Aust N Z J Obstet Gynaecol 1987;27:1–5.

29. Davidson KM et al. Acute fatty liver of pregnancy in triplet gestation. Obstet Gynecol 1998;91:806–8.

30. Malone FD et al. Maternal morbidity associated with triplet pregnancy. Am J Perinatol 1998;15:73–7.

31. Chauhan S, Scardo J, Hayes E, Abuhamad A, Berghella V. Twins: prevalence, problems, and preterm births. *Am J Obstet Gynecol* 2010;203(4):305-315.

32. Lewi L et al. Monochorionic diamniotic twin pregnancies: natural history and risk stratification. *Fetal Diagn Ther* 2010;27:121-133.

33. Chalouhi GE et al. Laser therapy for twin-to-twin transfusion syndrome (TTTS). *Prenat Diagn* 2011;31:637-646.

34. Haverkamp F, Lex C, Hanisch C, Fahnenstich H, Zerres K. Neurodevelopmental risks in twin-to-twin transfusion syndrome: preliminary findings. *Eur J Paediatr Neurol* 2001;5:21-27.

35. Lopriore E, Middeldorp JM, Oepkes D, Kanhai HH, Walther FJ, Vandenbussche FP. Twin anemia-polycythemia sequence in two monochorionic twin pairs without oligopolyhydramnios sequence. *Placenta* 2007;28:47-51.

36. Lewi L et al. Clinical outcome and placental characteristics of monochorionic diamniotic twin pairs with early- and late-onset discordant growth.*Am J Obstet Gynecol* 2008;199:511.

37. Lewi L et.Placental sharing, birthweight discordance, and vascular anastomoses in monochorionic diamniotic twin placentas. *Am J Obstet Gynecol* 2007;197:587.

38. Dickinson J. Monoamniotic twin pregnancy: a review of contemporary practice. *Aust N Z J Obstet Gynecol* 2005;45:474-478.

39. Arabin L et al. Early prenatal diagnosis of cord entanglement in monoamniotic multiple pregnancies. *Ultrasound Obstet Gynecol* 1999;13:140-142.

40. Royal College of Obstetricians and Gynaecologists. Greentop Guideline No. 51. Management of monochorionic twin pregnancy, December 2008.

41. Carlos J Echeverry. Multifetal gestation, Practical guide to high risk pregnancy and delivery, Fernando arias 3 rd edition pages 293 – 322.

42. Moore TR, Gale S, Benirschke K. Perinatal outcome of forty-nine pregnancies complicated by acardiac twinning. *Am J Obstet Gynecol* 1990;163:907-12.

43. Schinzel et al, Monozygotic twinning and structural defects. J Pediatr 95:951, 1979.

44. Bianco AT et al.The clinical outcome of preterm premature rupture of membranes in twin versus singleton pregnancies. Am J Perinatol 1996;13: 135 -138.

45. Assuncao RA et al. Perinatal outcome of twin pregnancies delivered in a teaching hospital.Rev assoc Med Bras 2010; 56(4): 447 -51

46. Azubike et al. Multiple births in Igbo women; Br J Obstet Gynaecol. 1982

47. Summera alsam et al. A comparative study between MC and DC twins to assess perinatal outcome.

48. Chowdhury et al.Maternal complications in twin pregnancies.Mymensingh Med J.2011;20(1):83-7.

49. PA Hatkar et al.Perinatal outcome of twins in relation to chorionicity JPGM 19999;vol 45;Issue 2, Page 33-7.

50. Naushaba Rizwan et al. Maternal morbidity and perinatal outcome with twin pregnancy. J Ayub Med Coll Abbottabad 2010;22(2)

51. Svetlana et al. Stillbirth and neonatal mortality in monochorionic and dichorionic twins:a population- based study. Hum. Reprod.(2011)doi.10.1093

52. S.V.Glinianaia et al. Congenital anomalies in twins: a register based study Hum. Reprod.(2008) 23 (6): 1306 – 11.

53. Dominigues et al. Chorionicity in twin pregnancies: impact upon perinatal results .Acta Obstet Ginecol Port 2007;1(4):163-166.

PERINATAL OUTCOME IN TWIN PREGNANCY ACCORDING TO CHORIONICITY - PROFORMA

NAME: ADDRESS:

AGE: I.P NO:

FAMILY HISTORY:

GRAVIDA:

MODE OF CONCEPTION:

MATERNAL RISK FACTORS:

- Anemia
- Gestational hypertension & Preeclampsia
- GDM
- Placenta previa

GESTATIONAL AGE AT DELIVERY:

PRETERM/PROM:

MODE OF DELIVERY:

PPH:

CHORIONICITY:

- 1 st trimester
- After delivery

IUD/STILL BIRTH:

TWIN A

TWIN B

BIRTH WEIGHT:

>2.5 kg

<2.5 kg

< 1.5 kg

CONGENITAL ANOMALIES:

TWIN A

TWIN B

IUGR:

TWIN A

TWIN B

APGAR SCORE < 7 AT 5 MINS:

TWIN A

TWIN B

DISCORDANT TWINS:

LIVE BIRTH:

TWIN A

TWIN B

NICU ADMISSION:

TWIN A

TWIN B

CAUSE OF NICU ADMISSION:

TWIN A
TWIN B

NEONATAL DEATH:

TWIN A

TWIN B

CAUSE OF DEATH:

TWIN A

TWIN B

LIVE BABY ON DISCHARGE:

TWIN A

TWIN B

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI -3

EC RegNo :ECR/270/Inst./TN/2013
Telephone No : 044 25305301
Fax : 044 25363970

CERTIFICATE OF APPROVAL

To
Dr.G.Arunadevi,
PG in MS(OG),
Institute of Social Obstetrics,
Govt. Kasturi Bai Gandhi Hospital, Madras Medical College, Chennai-3.

Dear G.Aurnadevi

The Institutional Ethics committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled "Perinatal outcome in Twin pregnancy according to chorionicity" No.05072013.

The following members of Ethics Committee were present in the meeting held on 02.07.2013 conducted at Madras Medical College, Chennai -3.

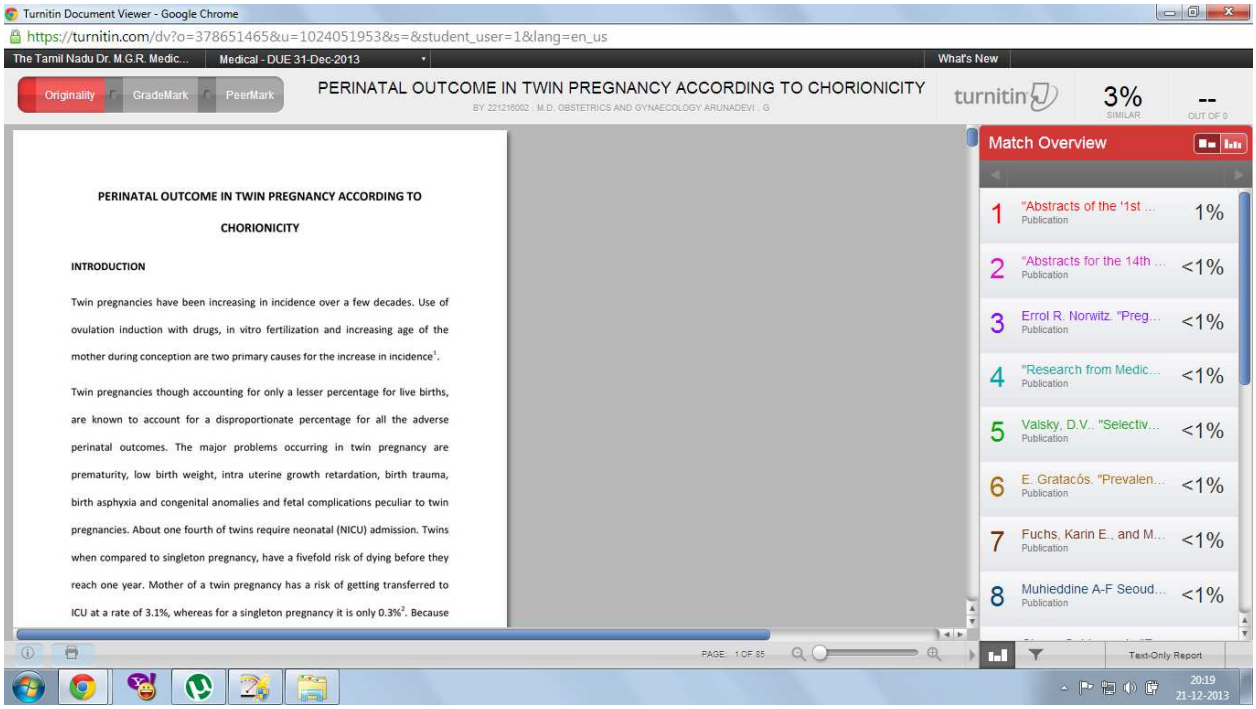
1. Dr.G.SivaKumar, MS FICS FAIS --- Chairperson
2. Prof. R. Nandhini MD -- Member Secretary
   Director, Instt. of Pharmacology ,MMC, Ch-3
3. Prof. Shyamraj MD -- Member
   Director i/c , Instt. of Biochemistry , MMC, Ch-3
4. Prof. P. Karkuzhali. MD -- Member
   Prof., Instt. of Pathology, MMC, Ch-3
5. Prof. Kalai Selvi -- Member
   Prof of Pharmacology, MMC, Ch-3
6. Prof. Siva Subramanian, -- Member
   Director, Instt. of Internal Medicine, MMC, Ch-3
7. Thiru. S. Govindsamy. BABL -- Lawyer
8. Tmt. Arnold Saulina MA MSW -- Social Scientist

We approve the proposal to be conducted in its presented form.

Sd/ Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

R Nadini 12/7/13
Member Secretary, Ethics Committee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PERINATAL OUTCOME IN TWIN PREGNANCY ACCORDING TO
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INTRODUCTION
Twin pregnancies have been increasing in incidence over a few decades. Use of ovulation induction with drugs, in vitro fertilization and increasing age of the mother during conception are two primary causes for the increase in incidence[1].
Twin pregnancies though accounting for only a lesser percentage for live births, are known to account for a disproportionate percentage for all the adverse perinatal outcomes. The major problems occurring in twin pregnancy are prematurity, low birth weight, intra uterine growth retardation, birth trauma, birth asphyxia and congenital anomalies and fetal complications peculiar to twin pregnancies. About one fourth of twins require neonatal (NICU) admission. Twins when compared to singleton pregnancy, have a fivefold risk of dying before they reach one year. Mother of a twin pregnancy has a risk of getting transferred to ICU at a rate of 3.1%, whereas for a singleton pregnancy it is only 0.3%[2]. Because
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PERINATAL OUTCOME IN TWIN PREGNANCY ACCORDING TO CHORIONICITY INTRODUCTION Twin pregnancies have been increasing in incidence over a few decades. Use of ovulation induction with drugs, in vitro fertilization and increasing age of the mother during conception are two primary causes for the increase in incidence1. Twin pregnancies though accounting for only a lesser percentage for live births, are known to account for a disproportionate percentage for all the adverse perinatal outcomes. The major problems occurring in twin pregnancy are prematurity, low birth weight, intra uterine growth retardation, birth trauma, birth asphyxia and congenital anomalies and fetal complications peculiar to twin...

PERINATAL OUTCOME IN TWIN PREGNANCY ACCORDING TO CHORIONICITY

Abstract

This study was a prospective study conducted on 100 twin pregnancies. The study was conducted at the Institute of Social Obstetrics, Government Kasturba Gandhi Hospital between December 2012 to November 2013.

Aim

To assess the morbidity and mortality of twins in relation to chorionicity and to analyse the factors responsible for that.

Material and methods

The information pertaining to the study like age, parity, gravida, residence, family history of twin pregnancy was obtained from the patients. Chorionicity was assessed using ultrasound and placental examination. The perinatal outcome was recorded in terms of gestational age at delivery (28 – 30 weeks, 31-33, 34-37, > 37 weeks), mode of delivery (Caesarian section/vaginal delivery/combined/outlet forceps/vacuum), Apgar score at 0 and 5 mins, birth weight(> 2500 gms, 2500 – 1500 gms, < 1500 gms), gender, dead/still/alive, babies getting admitted to NICU, number of days in ICU, and the final outcome of the babies, in terms of whether the baby got discharged in good condition or expired.

Results

Among the 100 twin pregnancies, 62% were dichorionic diamniotic, 34% were monochorionic diamniotic and 4% were monochorionic and monoamniotic.

Primigravidas constituted for 43% of pregnancies and multigravidas constituted for 57%. In both mono and dichorionic pregnancies the common age group was 25 – 29 years.

Of the maternal antepartum complications, preeclampsia ranked first which was present in about 25%, anemia occurred in 20%, GDM in 4% and APH in 3% and hydramnios in 1% out of the 100 twin deliveries. The mean gestational age of delivery for MCDA was 33 weeks compared to DCDA which was 34 weeks. PPH was present in 21% of MCDA and 16% of DCDA.

Of the specific complication in relation to chorionicity, the incidence of IUD, selective IUGR , congenital anomalies ,discordant growth were more in MCDA. Still birth rate was equal in both. The fetal outcome in terms of NICU admission, number of twins expired, apgar score <7, number of babies discharged alive showed statistical significance between MCDA and DCDA and more adverse outcome was noted in MCDA.

The most common cause for neonatal death for both MCDA and DCDA in our study was RDS and sepsis secondary to prematurity, followed by very low birth weight.

Conclusion

Since the incidence of maternal and fetal adverse outcome are increased significantly in twin pregnancies, it is highly advisable to determine the chorionicity at 11-14 weeks of gestation as each type of placentation carries different prognosis and morbidity. Regular ultrasound study and if needed Doppler study for the growth and wellbeing of the twins particularly monochorionic twins is mandatory. Assessment of chorionicity helps in the management of discordant growth, twin to twin transfusion, feasibility of multifetal reduction and management of other complications.

Keywords

Twin pregnancy, chorionicity, ultrasound, neonatal morbidity and mortality.

S.No.	Name	Age	I.P.No.	Gravida	Mode of Conception	Family History	Maternal Risk Factors	Gest. Age at Delivery	Preterm Prom	Mode of Delivery	Presentation	PPH	Chorionicity	IUD		Still Birth	
														A	B	A	B
1	Maheswari	26	14298	G3P1L1	Spont	-	-	37 wks	-	Vaginal	Vx Vx	-	DCDA	-	-	-	-
2	Pavithra	24	12432	Primi	Spont	P	-	35 wks	Preterm	LSCS	Breech Vx	-	MCDA	-	-	-	-
3	Vijitha	29	15727	G2P1L1	Spont	-	Gest. HT	30 wks	-	LSCS	Breech Vx	-	DCDA	-	-	-	-
4	Mohiba	26	16177	Primi	Spont	-	Preeclampsia	35 wks	Preterm	LSCS	Breech Vx	-	MCDA	-	-	-	-
5	Layeekha	23	12164	Primi	Spont	P	severe preeclampsia	33 wks	Preterm Pprom	Vaginal	Vx Vx	P	DCDA	-	-	-	-
6	Muthumari	29	17437	G2A1	Induced(IUI)	P	-	34 wks	Prom	Vaginal	Vx Transverse	-	MCDA	-	-	-	-
7	Manjula	26	17220	G2A1	Induced(IUI)	-	-	33 wks	Preterm Pprom	Vaginal	Vx Vx	-	DCDA	-	-	-	-
8	Rekha	24	16718	G3P1L1	Spont	-	-	37 wks	-	LSCS	Transverse Vx	-	DCDA	-	-	-	-
9	Saraswathy	26	17082	G2P1L1	Spont	P	-	38 wks	-	LSCS	Vx Vx	-	MCDA	-	-	-	-
10	Malliga	27	15116	G2P1L1	Induced(Dru)	-	-	36 wks	Preterm	LSCS	Vx Breech	-	DCDA	-	-	-	-
11	Bhavani	22	16468	Primi	Spont	P	-	30 wks	Preterm	Vaginal	Vx Breech	-	MCDA	-	P	-	-
12	Ezhilarasi	23	7409	Primi	Induced(Dru)	-	-	37 wks	Prom	Outlet Vacuum	Vx Vx	-	DCDA	-	-	-	-
13	Malathi	26	6842	G2P1L1	Induced(Dru)	-	-	32 wks	Pprom	LSCS	Vx Breech	-	MCDA	-	-	-	-

DRU - Drugs, P - Present, Vx - vertex

S.No.	Birth Weight >2.5 KG		Birth Weight <2.5 KG		Very Low Birth Weight <1.5 KG		Congenital Anomalies		IUGR		APGAR SCORE at 5 mins		Discordant Twins	Time Interval Delivering Mins	Live Birth on Delivery		NICU Admission		Neonatal Death		Cause of Death		Live Baby on Discharge		No of Days Nicu Admission	
	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	>20%		TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B
1	3.2kg	2.7kg	-	-	-	-	-	-	-	-	8/10	8/10	-	5	A	A	-	-	-	-	-	-	A Boy	A Girl		
2	-	-	1.97	-	-	1.48	-	-	-	-	8/10	8/10	P	2	A	A	RDS	RDS	-	-	-	-	A Girl	A Girl	15	20
3	-	-	2.3	2.1	-	-	-	-	-	-	7/10	7/10	-	1	A	A	-	-	-	-	-	-	A Boy	A Boy	0	nil
4	-	-	1.7	-	-	1.4	-	-	-	-	8/10	3/10	-	1	A	A	LBW	LBW & RDS	-	-	-	-	A Girl	D Girl	11	nil
5	-	-	1.96	1.93	-	-	-	-	-	-	7/10	7/10	-	10	A	A	Preterm/LBW	Preterm/LBW	-	-	-	-	A Girl	A Boy	7	9
6	-	-	-	-	1.5	1.35	-	-	-	-	8/10	2/10	-	30	A	A	RDS	RDS	-	-	-	RDS & Sepsis	A Boy	D Boy	12	nil
7	-	-	-	-	1.3	1.5	-	-	-	-	4/10	4/10	-	5	A	A	VLBW/RDS	VLBW/RDS	RDS & Sepsis	RDS & Sepsis	-	-	D Boy	D Boy	2	3
8	2.7	2.6	-	-	-	-	-	-	-	-	8/10	8/10	-	1	A	A	-	-	-	-	-	-	A Boy	A Girl	nil	nil
9	2.6	2.6	-	-	-	-	-	-	-	-	8/10	8/10	-	1	A	A	-	-	-	-	-	-	A Boy	A Boy	nil	nil
10	-	-	2.05	1.7	-	-	-	-	-	-	8/10	6/10	-	1	A	A	RDS	RDS	-	D	-	NEC	A Boy	D Boy	8	nil
11	-	-	-	-	1.015	500	-	-	-	P	7/10	0/10	P	20	A	D	RDS & LBW	-	-	-	-	-	A Girl	D Boy	17	nil
12	2.6	2.6	-	-	-	-	-	-	-	-	8/10	2/10	-	35	A	A	RDS	RDS	-	D	-	Birth Asp.	A Boy	D Girl	5	nil
13	-	-	1.65	1.62	-	-	-	-	-	-	8/10	9/10	-	1	A	A	RDS	RDS	D	D	Sepsis	Sepsis	D Boy	D Boy	nil	nil

P - Present, VLBW - Very low birth weight, RDS - Respiratory distress syndrome, LBW - Low birth weight, A - Alive, D - Death NEC - necrotising enterocolitis. Birth asp.- birth asphyxia

S.No.	Name	Age	I.P.No.	Gravida	Mode of Conception	Family History	Maternal Risk Factors	Gest. Age at Delivery	Preterm Prom	Mode of Delivery	Presentation	PPH	Chorionicity	IUD		Still Birth	
														A	B	A	B
14	Soundaravalli	27	6839	G3P1L1A1	Spont	-	-	37	Prom	Vaginal	Vx Vx	+	DCDA	-	-	-	-
15	Kanimozhi	28	8148	G2P1L1	Spont	-	-	35	Prom	LSCS	Breech Vx	-	DCDA	-	-	-	-
16	Gayathri	30	8050	G2P1L1	Spont	P	Gest.HT	32	Pprom	Vaginal	Vx Vx	-	MCDA	-	-	-	-
17	Parvathy	25	6127	G2P1L1	Spont	-	Preclamptic	37	-	Vaginal	Breech Vx	-	MCDA	-	-	-	-
18	Seethalakshmi	30	7207	G4P2L1A1	Spont	-	Anemia	38	-	Vaginal	Vx Vx	-	MCDA	-	-	-	-
19	Shakila	19	8696	Primi	Spont	-	Gest.HT	36	-	Vaginal	Breech Vx	-	MCDA	-	P	-	-
20	Rajeshwari	24	9242	G2P1L1	Spont	-	-	36	-	Vaginal	Breech Vx	-	DCDA	-	-	-	-
21	Poorani	27	11249	G4P2L1A1	Induced(Dru)	-	Severe Preclamptic	36	-	LSCS	Breech Transverse	-	MCDA	-	-	-	-
22	Malarvizhi	29	11903	Primi	Induced(Dru)	P	Gest.HT	35	Preterm	Vaginal	Vx Vx	-	DCDA	-	-	-	-
23	Naagiya	27	11972	G3P1L1A1	Spont	-	-	37	-	LSCS	Breech Vx	-	MCDA	-	-	-	-
24	Rigwana	24	8929	G2P1L1	Spont	-	-	37	-	Vaginal	Vx Vx	-	DCDA	-	-	-	-
25	Umamaheshwari	26	4334	Primi	Spont	-	-	37	-	LSCS	Breech Vx	-	DCDA	-	-	-	-

MCDA- monochorionic diamniotic, DCDA - dichorionic diamniotic, Gest. HT - gestational hypertension, Dru - drugs, P - present, Vx - verte›

S.No.	Birth Weight >2.5 KG		Birth Weight <2.5 KG		Very Low Birth Weight <1.5 KG		Congenital Anomalies		IUGR		APGAR SCORE at 5 mins		Discordant Twins	Time Interval Delivering Mins	Live Birth on Delivery		NICU Admission		Neonatal Death		Cause of Death		Live Baby on Discharge		No of Days Nicu Admission	
	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	>20%		TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B
14	2.5	-	-	2.4	-	-	-	-	-	-	9/10	9/10	-	10	A	A	-	-	-	-	-	-	A Girl	A Girl	nil	nil
15	-	-	1.5	1.8	-	-	-	-	-	-	8/10	5/10	-	1	A	A	RDS	RDS	D	-	HMD	-	D Girl	A Girl	16	nil
16	-	-	-	-	1.4	1.2	-	-	-	-	5/10	5/10	-	15	A	A	LBW/prem aturity	LBW/prem aturity	D	D	RDS Sepsis	RDS & Sepsis	D Boy	D Boy	nil	nil
17	3.1	-	-	2	-	-	-	-	-	P	8/10	7/10	P	10	A	A	-	-	-	-	-	-	A Boy	A Boy	nil	nil
18	2.5	-	-	2.4	-	-	-	-	-	-	9/10	6/10	-	20	A	A	-	Birth Asphyxia	-	-	-	-	A Boy	A Boy	nil	4
19	-	-	2.3	300g	-	-	-	-	-	-	9/10	0/10	P	30	A	D	-	-	-	-	-	-	A Girl	D Girl	nil	nil
20	-	-	-	2.2	1.1	-	-	-	-	-	4/10	7/10	P	22	D	A	LBW	-	D	-	RDS	-	D Boy	A Boy	nil	nil
21	-	-	2.4	2	-	-	-	-	-	-	8/10	8/10	-	1	A	A	-	LBW	-	-	-	-	A Boy	A Girl	nil	8
22	-	-	-	-	1.8	1.85	-	-	-	-	8/10	9/10	-	10	A	A	LBW	LBW	-	-	-	-	A Boy	A Boy	17	12
23	2.9	-	-	2.4	-	-	-	-	-	-	7/10	9/10	-	1	A	A	-	-	-	-	-	-	A Boy	A Boy	nil	nil
24	-	2.8	2	-	-	-	-	-	-	-	7/10	9/10	P	15	A	A	-	-	-	-	-	-	A Boy	A Girl	nil	nil
25	-	-	2.3	2.4	-	-	-	-	-	-	8/10	8/10	-	2	A	A	-	-	-	-	-	-	A Girl	A Girl	nil	nil

P - present, LBW - low birth weight, RDS - respiratory distress syndrome, A - alive, D - death

S.No.	Name	Age	I.P.No.	Gravida	Mode of Conception	Family History	Maternal Risk Factors	Gest. Age at Delivery	Preterm Prom	Mode of Delivery	Presentation	PPH	Chorionicity	IUD		Still Birth	
														A	B	A	B
26	Anandhi	27	5944	Primi	Spont	-	-	34	Preterm	LSCS	Transerve	-	MCDA	-	-	-	-
27	Sharebanu	29	5885	G2A1	Spont	-	Preeclamptic GDM	36	-	LSCS	Transerve Breech	PPH	DCDA	-	-	-	-
28	Kalaimathi	18	10539	Primi	Spont	-	-	32	Pprom	Vaginal	Breech Breech	-	DCDA	-	-	-	-
29	Malliga	19	10520	Primi	Spont	-	-	36	-	Vaginal	Vx Breech	-	DCDA	-	-	-	-
30	Malarkodi	27	11505	G2P1L1	Spont	-	-	37	-	LSCS	Vx Breech	-	DCDA	-	-	-	-
31	Kumari	29	13110	G2P1L1	Spont	P	-	32	Preterm	LSCS	Vx Vx	PPH	DCDA	-	-	-	-
32	Anadhi	29	5419	G4P2L1A1	Spont	-	-	35	Preterm	LSCS	Breech Vx	-	DCDA	-	-	-	-
33	Ammu	30	5532	G4P2L1A1	Spont	-	Anemia	37	-	LSCS	Breech Breech	-	DCDA	-	-	-	-
34	Anandhi	36	5671	G6P3L3A2	Spont	-	Preeclamptic	37	-	Vaginal	Vx Breech	-	DCDA	-	-	-	-
35	Alamelu	25	17944	G2P1L1	Spont	-	-	38	-	LSCS	Vx Breech	-	MCDA	-	-	-	-
36	Gracy	27	16841	G3P2L2	Spont	P	GDM	38	-	LSCS	Vx Vx	-	DCDA	-	-	-	-
37	Usha	28	17711	G3P1L1	Spont	P	-	35	Prom	LSCS	Vx Breech	-	DCDA	-	-	-	-

P - present, PPH - postpartum hemorrhage, GDM - gestational diabetes mellitus, MCDA - monochorionic diamniotic, DCDA - dichorionic diamniotic

S.No.	Birth Weight >2.5 KG		Birth Weight <2.5 KG		Very Low Birth Weight <1.5 KG		Congenital Anomalies		IUGR		APGAR SCORE at 5 mins		Discordant Twins	Time Interval Delivering Mins	Live Birth on Delivery		NICU Admission		Neonatal Death		Cause of Death		Live Baby on Discharge		No of Days Nicu Admission	
	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	>20%		TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B
26	-	-	1.9	2.3	-	-	-	-	-	-	8/10	9/10	-	2	A	A	Preterm/LBW	-	-	-	-	-	A Girl	A Girl	8	nil
27	3.07	-	-	2.46	-	-	-	-	-	-	9/10	9/10	-	2	A	A	-	-	-		-	-	A Girl	A Girl	nil	nil
28	-	-	-	-	1.145	1.155	-	-	-	-	6/10	6/10	-	15	A	A	VLBW/ RDS	VLBW/RDS	D	D	RDS	RDS	D Boy	D Girl	3	2
29	-	-	2.27	2.06	-	-	-	-	-	-	8/10	5/10	-	30	A	A	-	Birth Asp.	-	D	-	Birth Asp.	A Boy	D Girl	nil	1
30	2.36	-	-	2.15	-	-	-	-	-	-	8/10	9/10	-	1	A	A	-	-	-	-	-	-	A Girl	A Girl	nil	nil
31	-	-	1.8	1.9	-	-	-	-	-	-	5/10	4/10	-	2	A	A	preterm	preterm	-	D	-	RDS	A Boy	D Boy	15	5
32	-	-	2.1	1.8	-	-	-	-	-	-	8/10	8/10	-	1	A	A	-	LBW RDS	-	-	-	-	A Girl	A Boy	nil	nil
33	-	-	2.3	2.02	-	-	-	-	-	-	7/10	8/10	-	1	A	A	-	-	-	-	-	-	A Boy	A Boy	nil	nil
34	2.5	2.5	-	-	-	-	-	-	-	-	9/10	9/10	-	17	A	A	-	-	-	-	-	-	A Boy	A Boy	nil	nil
35	2.2	3.1	-	-	-	-	-	-	-	-	8/10	8/10	P	1	A	A	-	-	-	-	-	-	A Boy	A Boy	nil	nil
36	2.5	-	-	2.4	-	-	-	-	-	-	8/10	8/10	-	1	A	A	-	-	-	-	-	-	A Girl	A boy	nil	nil
37	-	-	1.95	2.1	-	-	-	-	-	-	9/10	7/10	-	-	A	A	LBW	-	-	-	-	-	A Boy	A Boy	4	nil

VLBW - very low birth weight, Birth asp. - birth asphyxia, P- present, A - alive, D - death, LBW - low birth weight, RDS - respiratory distress syndrome

S.No.	Name	Age	I.P.No.	Gravida	Mode of Conception	Family History	Maternal Risk Factors	Gest. Age at Delivery	Preterm Prom	Mode of Delivery	Presentation	PPH	Chorionicity	IUD		Still Birth	
														A	B	A	B
38	Subha	31	6664	G3P1L1A1	Spont	-	-	34	Prom	LSCS	Vx Transverse	-	MCDA	-	-	-	-
39	Lakshmi	26	13252	Primi	Spont	-	Hypothy	34	Preterm	LSCS	Breech Breech	-	MCDA	-	-	-	-
40	Vijayalakshmi	20	4748	G2P1L1	Spont	-	-	37	-	LSCS	Vx Vx	-	MCMA	-	-	-	-
41	Rajitham	22	4625	G2P1L1	Induced(Dru)	-	-	36	-	LSCS	Vx Transverse	-	MCMA	-	-	-	-
42	Radha	20	4699	G2P1L1	Spont	-	anemia	37	Prom	LSCS	Vx Transverse	-	DCDA	-	-	-	-
43	Mahalakshmi	22	4084	G2A1	Induced(Dru)	P	-	33	Pprom	LSCS	Breech Breech	+	MCDA	-	-	-	-
44	Vatchala	30	3150	Primi	Spont	-	Preclamptic	37	-	LSCS	Vx Vx	-	MCDA	-	-	-	-
45	Moneka	24	4684	Primi	Spont	-	Preclamptic	37	-	LSCS	Vx Breech	-	DCDA	-	-	-	-
46	Deepa	19	3445	Primi	Spont	-	-	34	-	Lab N	Vx Vx	-	DCDA	-	-	-	P
47	Priya	24	3585	G3P1L1A1	Spont	-	GDM	37	-	Vaginal	Vx Vx	-	DCDA	-	-	-	-
48	Bhakya	24	2516	Primi	Spont	-	-	37	-	Vaginal	Vx Breech	-	MCMA	P(cord)	-	-	-
49	Shamsath	20	3825	Primi	Spont	-	-	35	-	LSCS	Transverse Vaginal	-	DCDA	-	-	-	-

P - present, Dru - drugs, MCMA - monochorionic monoamniotic, cord - cord entanglement, hypothy - hypothyroidism, MCDA - monochorionic diamniotic, DCDA - dichorionic diamniotic, Vx- vertex

S.No.	Birth Weight >2.5 KG		Birth Weight <2.5 KG		Very Low Birth Weight <1.5 KG		Congenital Anomalies		IUGR		APGAR SCORE at 5 mins		Discordant Twins	Time Interval Delivering Mins	Live Birth on Delivery		NICU Admission		Neonatal Death		Cause of Death		Live Baby on Discharge		No of Days Nicu Admission	
	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	>20%		TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B
38	-	-	1.9	1.95	-	-	-	Spina Bifida	-	-	7/10	7/10	-	1	A	A	LBW/RDS	LBW/RDS	-	-	-	-	A Girl	A Girl	7	8
39	-	-	1.7	7.2	-	-	-	Genu recurvatum	-	-	8/10	8/10	-	1	A	A	LBW/HMD	LBW/HMD	-	-	-	-	A Boy	A Boy	14	18
40	-	-	2.1	1.63	-	-	-	-	-	P	8/10	4/10	P	2	A	A	-	Hyperbilirubinemia	-	D	A	HMD	A Boy	A Boy	nil	4
41	-	-	1.8	2	-	-	-	-	-	-	8/10	9/10	-	1	A	A	LBW	-	-	-	-	-	A Boy	A Boy	14	nil
42	-	-	2	1.75	-	-	-	-	-	-	8/10	7/10	-	2	A	A	-	LBW & RDS	-	-	-	-	A Girl	A Girl	nil	10
43	-	-	1.67	1.96	-	-	-	-	-	-	9/10	5/10	-	1	A	A	Preterm/LBW	Preterm/LBW	D	A	sepsis	-	D Girl	A Girl	nil	8
44	2.7	2.6	-	-	-	-	-	Single Umb. Artery	-	-	9/10	9/10	-	1	A	A	-	Single umbilical artery	-	-	-	-	A Boy	A Boy	nil	4
45	2.7	-	-	2.3	-	-	-	-	-	-	8/10	8/10	-	1	A	A	-	-	-	-	-	-	A Boy	A Girl	nil	nil
46	-	-	1.7	1.6	-	-	-	-	-	-	8/10	0/10	-	36	A	D	-	-	-	-	-	-	A Boy	D Boy	nil	nil
47	2.9	2.9	-	-	-	-	-	-	-	-	8/10	8/10	-	16	A	A	-	-	-	-	-	-	A Boy	A Boy	nil	nil
48	-	2.78	-	-	-	-	-	-	-	-	8/10	8/10	-	15	D	A	-	-	-	-	-	-	D Boy	A Boy	nil	nil
49	-	-	1.76	1.9	-	-	PDA	-	-	-	8/10	8/10	-	1	A	A	LBW	LBW	-	-	-	-	A Boy	A Girl	11	7

A - alive, D - death, HMD - hyaline membrane disease

Single umb. Artery - single umbilical artery, P - present, LBW - low birth weight, RDS - respiratory distress syndrome, HMD - hy

S.No.	Name	Age	I.P.No.	Gravida	Mode of Conception	Family History	Maternal Risk Factors	Gest. Age at Delivery	Preterm Prom	Mode of Delivery	Presentation	PPH	Chorionicity	IUD		Still Birth	
														A	B	A	B
50	Sathya	29	6715	Primi	Spont	-	-	35	Preterm	LSCS	Breech Vx	P	DCDA	-	-	-	-
51	Shambath	30	3825	Primi	Spont	P	-	35	Preterm	LSCS	Transverse Vx	-	DCDA	-	-	-	-
52	Priya	33	3668	Primi	Spont	-	-	36	-	Vaginal	Vx Vx	-	DCDA	-	-	-	-
53	Geetha	33	1077	G2P1L1	Spont	-	Abruption	37	-	LSCS	Vx Vx	-	DCDA	-	-	-	-
54	Ajirabanu	21	907	G4P1L1A2	Spont	-	Anemia	35		LSCS	Breech Breech	P	MCDA	P	-	-	-
55	Asma	27	12203	G5P2L1A2	Spont	-	Gest HT	38	-	LSCS	Transverse Vx	-	DCDA	-	-	-	-
56	Shakira	23	930	Primi	Spont	-	Preeclampsia	33	Preterm Pprom	LSCS	Breech Vx	-	DCDA	-	-	-	-
57	Sumithra	22	1544	Primi	Spont	-	GDM	36	-	LSCS	Vx Vx	-	DCDA	-	-	-	-
58	Megala	24	15984	Primi	Spont	-	-	32	Preterm Pprom	LSCS	Vx Vx	-	MCDA	-	-	-	-
59	Subha	31	6664	G3P1L1A1	Spont	-	Anemia	34	Prom	LSCS	Vx Vx	-	MCDA	-	-	-	-

Gest HT - Gestational hypertension, GDM - gestational diabetes mellitus, Vx - vertex, DCDA - dichorionic diamniotic, MCDA - monochorionic diamniotic, P - present,

S.No.	Birth Weight >2.5 KG		Birth Weight <2.5 KG		Very Low Birth Weight <1.5 KG		Congenital Anomalies		IUGR		APGAR SCORE at 5 mins		Discordant Twins >20%		Time Interval Delivering Mins	Live Birth on Delivery		NICU Admission		Neonatal Death		Cause of Death		Live Baby on Discharge		No of Days Nicu Admission	
	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B		TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B
50	-	2.7	1.93	-	-	-	-	-	-	-	8/10	8/10.	-	P	1	A	A	-	-	-	-	-	-	A Boy	A Girl	nil	nil
51	-	-	1.76	1.9	-	-	-	-	-	-	8/10	8/10.	-	-	1	A	A	Preterm/RDS	Preterm/RDS	-	-	0	HMD	A Boy	A Girl	18	16
52	2.5	2.38	-	-	-	-	-	-	-	-	6/10	7/10	-	-	5	A	A	-	-	-	-	-	-	A Girl	A Boy	nil	nil
53	-	-	2.45	2.25	-	-	-	-	-	-	7/10	8/10	-	-	1	D	A	-	-	-	-	-	-	A Boy	A Boy	nil	nil
54	-	-	-	-	1.9	2.3	-	-	-	-	0/10	8/10	-	-	1	A	A	-	-	-	-	-	-	D Boy	A Boy	nil	nil
55	2.96	2.96	-	-	-	-	-	-	-	-			-	-	1	A	A	-	-	-	-	-	-	A boy	A boy	nil	nil
56		-	-	-	1.2	1.2	-	-	-	-	6/10	6/10	-	-	1	A	A	LBW & RDS	LBW & RDS	D	-	RDS	RDS	D Girl	D Girl	19	24
57	2.6	-	-	2	-	-	-	-	-	P	9/10	9/10	-	P	1	A	A	-	Hyperbilirubinemia	-	-	-	-	A Girl	A Boy	nil	8
58	-	-	-	-	1.2	1.9	cleft palate	cleft palate	P	-	7/10	7/10	P	-	1	A	A	RDS	LBW	-	-	-	-	A Girl	A Girl	18	5
59		-	1.9	1.95	-	-	-	spina bifida	-	-	9/10	9/10	-	-	1	A	A	LBW	LBW	-	-	-	-	A Girl	A Girl	16	16

P -present, A -alive, RDS -respiratory distress syndrome, LBW - low birth weight,

S.No.	Name	Age	I.P.No.	Gravida	Mode of Conception	Family History	Maternal Risk Factors	Gest. Age at Delivery	Preterm Prom	Mode of Delivery	Presentation	PPH	Chorionicity	IUD		Still Birth	
														A	B	A	B
60	Amala	25	18880	Primi	Spont	-	Placenta previa	33	Preterm	LSCS	Vx Vx	P	DCDA	-	-	-	-
61	Muthumeena	33	18117	G3P1L1	Spont	-	-	37		LSCS	Vx Vx	-	MCDA	-	-	-	-
62	Shabina Begam	28	7658	G2P1L1	Spont	-	-	37	-	Vaginal	Vx Vx	-	MCDA	-	-	-	-
63	Gandhimathi	32	15748	G3P2L2	Spont	-	anemia	35	Preterm	Vaginal	Vx Breech	-	DCDA	-	-	-	-
64	Lavanya	23	19128	Primi	Induced(dru)	-	-	38		LSCS	Breech Vx	P	MCDA	-	-	-	-
65	Suganthi	27	19450	G2P1L1	Induced(dru)	-	-	36	-	LSCS	Vx Vx	-	DCDA	-	-	-	-
66	Revathi	26	1869	G2P1L1	Induced(dru)	-	Gest,HT	36	-	LSCS	Vx Vx	-	DCDA	-	-	-	-
67	Sameem	21	18214	Primi	Spont	-	Gest,HT	37	Prom	LSCS	Transverse Vx	-	DCDA	-	-	-	-
68	Shathi	27	18412	G2P1L1	Spont	-	-	34	Prom	LSCS	Breech Vx	-	DCDA	-	-	-	-

dru -drugs, Vx -vertex, P -present, DCDA -dichorionic diamniotic, MCDA - monochorionic diamniotic

S.No.	Birth Weight >2.5 KG		Birth Weight <2.5 KG		Very Low Birth Weight <1.5 KG		Congenital Anomalies		IUGR		APGAR SCORE at 5 mins		Discordant Twins >20%		Time Intervel Delivering Mins	Live Birth on Delivery		NICU Admission		Neonatal Death		Cause of Death		Live Baby on Discharge		No of Days Nicu Admission	
	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B		TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B
60	-		2.11	2.12	-	-	-	-	-	-	8/10	8/10	-	-	-	A	A	-	-	D		sepsis	-	D Girl	A Girl	5	nil
61	3.2	-	-	2.25	-	-	-	-	-	-	8/10	8/10	-	P	-	A	A	-	-	-	-	-	-	A Boy	A Boy	nil	nil
62	-	-	-	-	1.8	1.9	-	-	-	-	8/10	8/10	-	-	-	A	A	LBW	LBW/NEC	-	-	-	sepsis/pre maturity	A Girl	D Girl	11	7
63	2.6	-	-	2.3	-	-	-	-	-	-	8/10	8/10	-	-	-	A	A	-	-	-	-	-	-	A Boy	A Boy	nil	nil
64	2.6	3.3	-	-	-	-	single umbilical artery	-	-	-	8/10	8/10	-	-	-	A	A	single umbilical artery	-	-	-	-	-	A Boy	A Boy	8	nil
65	3.4	2.4	-	-	-	-	-	-	-	-	8/10	8/10	-	P	-	A	A	discordant growth	-	-	-	-	-	A Boy	A Boy	4	nil
66	-	-	-	-	1.4	0.9	-	-	-	-	4/10	4/10	-	-	-	A	A	Preterm	-	D	D	VLBW /IVH	sepsis/pre maturity	D Girl	D Girl	nil	nil
67	2.5	2.6	-	-	-	-	-	-	-	-	9/10	9/10	-	-	-	A	A	Seizures	-	-	-	-	-	A Girl	A Girl	4	nil
68	-	-	2.3	2.3	-	-	-	-	-	-	9/10	9/10	P	-	-	A	A	-	-	-	-	-	-	A Girl	A Boy	nil	nil

P - present, LBW - low birth weight, NEC - necrotising enterocolitis, A - alive, D -death, IVH - intraventricular hemorrhage

S.No.	Name	Age	I.P.No.	Gravida	Mode of Conception	Family History	Maternal Risk Factors	Gest. Age at Delivery	Preterm Prom	Mode of Delivery	Presentation	PPH	Chorionicity	IUD		Still Birth	
														A	B	A	B
69	Premit	32	13462	Primi	Spont	-	-	35	pprom	LSCS	VX Breech	-	MCDA	-	-	-	-
70	Violet Mary	28	12180	Primi	Spont	-	-	32		Vaginal	Vx Vx	-	MCDA	-	-	-	-
71	Vimala	21	20287	Primi	Induced(dru)	-		34	pprom	LSCS	Breech Vx	-	MCDA	-	-	-	-
72	Sasikala	24	19811	Primi	Induced(dru)	-	-	34		LSCS	Transverse Breech	-	DCDA	-	-	-	-
73	Kumari	20	17869	Primi	Spont	-	-	34		LSCS	Transverse Breech	-	DCDA	-	-	-	-
74	Bakiyalakshmi	20	9282	G2A1	Spont	-	preclampsia	32		Vaginal	Vx Vx	-	DCDA	-	-	-	-
75	Nochilli	24	10039	Primi	Spont	-	preclampsia	37	-	LSCS	Vx Vx	-	MCDA	-	-	-	-
76	Dhanalakshmi	30	12212	G2A1	Spont	-	hydramnios	34	-	Vaginal	Vx Vx	-	MCDA	-	-	-	cord prolapse
77	Vedhanagagi	32	17342	G2P1L1	Spont	-	-	33	-	LSCS	VX Breech	-	MCDA	-	-	-	-
78	Gayathri	30	1342	Primi	Spont	-	-	32	-	Vaginal	VX Breech	-	DCDA			-	-

dru -drugs, Vx - vertex, MCDA - monochorionic diamniotic, DCDA - dichorionic diamniotic

S.No.	Birth Weight >2.5 KG		Birth Weight <2.5 KG		Very Low Birth Weight <1.5 KG		Congenital Anomalies		IUGR		APGAR SCORE at 5 mins		Discordant Twins >20%		Time Interval Delivering Mins	Live Birth on Delivery		NICU Admission		Neonatal Death		Cause of Death		Live Baby on Discharge		No of Days Nicu Admission	
	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B		TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B
69	-	-	2.2	2	-	-	-	PDA	-	-	7/10	7/10	-	-	1 min	A	A	RDS	PDA PPHN		D		PDA PPHN	A girl	D Girl	10	nil
70	-	-	-	-	1.1	950g	-	-	-	-	6/10	2/10	-	-	10 mins	A	A	VLBW/RDS	VLBW/RDS	D	D	IVH	VLBW RDS	D Girl	D Girl	nil	nil
71	-	-	2.4	-	-	1.5	CTEV	-	-	P	7/10	4/10	-	P	1	A	A	-	VLBW/RDS			-	-	A girl	A girl	nil	11
72	2.7	-	-	2.1		-	-	-	-	-	8/10	8/10	-	-	2	A	A	-	-	-	-	-	-	A Boy	A girl	nil	nil
73	-	-	-	-	1.5	1.8	-	-	-	-	8/10	7/10	-	-	1	A	A	VLBW/RDS	VLBW/RDS	D		Preterm IVH	-	D Girl	A boy	nil	nil
74	2.5	2.6	-	-	-	-	-	-	-	-	8/10	7/10	-	-	0	A	A	Hyperbiliru binemia	-	-	-	-	-	A girl	A girl	17	nil
75	-	-	2.2	-	-	1.6	-	-	-	-	8/10	7/10	-	-	1	A	A	-	VLBW	-	-	-	-	A girl	A girl	nil	nil
76	-	-	2.2	-	-	1.9	-	-	-	-	8/10	0/10	-	-	10	A	D	-	-	-	-	-	-	A girl	D Girl	nil	nil
77	-	-	2	-	-	1.8	-	-	-	-	8/10	7/10	-	-	1	A	A	RDS	RDS	-	D	-	CHD	A girl	D Girl	7	nil
78	-	-	-	-	1.9	1.7	-	-	-	-	6/10	6/10	-	-	5	A	A	VLBW RDS	VLBW RDS	D	D	NEC	Sepsis	D Boy	D Boy	nil	nil

PDA - patent ductus arteriosus, CTEV - congenital talipus equino varus, PPHN - persistent pulmonary hypertension of newborn, VLBW - very low birth weight, RDS - respiratory distress syndrome, A -alive, D- death,
CHD- congenital heart disease, IVH - intraventricular hemorrhage, NEC - necrotising enterocolitis

S.No.	Name	Age	I.P.No.	Gravida	Mode of Conception	Family History	Maternal Risk Factors	Gest. Age at Delivery	Preterm Prom	Mode of Delivery	Presentation	PPH	Chorionicity	IUD		Still Birth	
														A	B	A	B
79	Poongodi	23	8681	G2A1	Spont	-	-	34	Preterm	Vaginal LSCS	Vx Breech	-	MCDA	-	-	-	-
80	Kumudha	29	7006	Primi	Spont	-	-	35	Preterm	Vaginal	Vx Vx	-	MCDA	-	-	-	-
81	Kumudha	18	7457	G2A1	Spont	-	-	37	Preterm	LSCS	Vx Breech	-	DCDA	-	Cord Entanglement	-	-
82	Devi	25	12963	Primi	Spont	-	Pre eclampsia	34	Preterm	Vacuum Extraction	Vx Vx	P	MCDA	-	-	-	-
83	Hajirabegam	23	11020	Primi	Spont	-	-	37	Preterm	LSCS	Vx Breech	-	DCDA	Thick Meconim	Thick Meconim	-	-
84	Anna Poorani	25	14813	G2P1L1	Spont	-	anemia	35	Preterm	Vaginal	Vx Breech	-	DCDA	-	-	-	-
85	Angayarkarasi	21	11176	G2P1L1	Spont	-	anemia	37	Preterm	LSCS	Vx Vx	-	DCDA	-	-	-	-
86	Emmaculate Mary	30	11279	G2P1L1	Spont	-	-	38	Preterm	LSCS	Breech Vx	-	DCDA	-	-	-	-
87	Valli	21	12120	G2P1L1	Spont	-	-	37	Preterm	Vaginal	Vx Vx	-	DCDA	-	-	-	P

P - present, MCDA - monochorionic diamniotic, DCDA - dichorionic diamniotic

S.No.	Birth Weight >2.5 KG		Birth Weight <2.5 KG		Very Low Birth Weight <1.5 KG		Congenital Anomalies		IUGR		APGAR SCORE at 5 mins		Discordant Twins >20%		Time Interval Delivering Mins	Live Birth on Delivery		NICU Admission		Neonatal Death		Cause of Death		Live Baby on Discharge		No of Days Nicu Admission	
	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B		TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B	TWIN A	TWIN B
79	-	-	1.67	1.9	-	-	-	-	-	-	8/10	5/10	-	-	30	A	A	LBW RDS	LBW/RDS	-	-	-	-	A Girl	A Girl	18	20
80	-	-	1.6	1.8	-	-	-	-	-	-	8/10	7/10	-	-	5	A	A	LBW RDS	LBW/RDS	D	D	LBW RDS	sepsis	D Girl	D Boy	nil	nil
81	-	-	2	2.4	-	-	-	-	-	-	8/10	8/10	-	-	1	A	A	Hyperbiliru benimia	-	-	-	-	-	A Boy	A Girl	nil	10
82	-	-	-	-	2.1	2.3	-	-	-	-	7/10.	7/10	-	-	7	A	A	RDS	RDS	-	-	-	-	A Boy	A Boy	nil	nil
83	2.4	2.3	-	-	-	-	-	-	-	-	0/10	0/10	-	-	25	D	D	-	-	-	-	-	-	D Girl	D Girl	nil	nil
84	-	-	2.2	2.4	-	-	-	-	-	-	.8/10	8/10	-	-	10	A	A	-	-	-	-	-	-	A Boy	A Girl	nil	nil
85	-	-	3.3	2.4	-	-	-	-	-	P	8/10	6/10	-	P	1	A	A	-	Birth asphyxia	-	-	-	-	A boy	A Girl	nil	6
86	-	-	2.5	2.5	-	-	-	-	-	-	8/10	8/10	-	-	1	A	A	-	-	-	-	-	-	A Girl	A Boy	nil	nil
87	2.6	-	2.2	2.2	-	-	-	-	-	-	8/10	0/10	-	-	15	A	D	Seizures	-	-	-	-	-	A Girl	D Boy	nil	nil

P - present, LBW - low birth weight, RDS - respiratory distress syndrome, A- alive, D -death

S.No.	Name	Age	I.P.No.	Gravida	Mode of Conception	Family History	Maternal Risk Factors	Gest. Age at Delivery	Preterm Prom	Mode of Delivery	Presentation	P	Chorionicity	IUD		Still Birth	
														A	B	A	B
88	Menaka	30	12120	G2P1L1	Spont	-	Gest HT	35	Preterm	Vaginal	Vx Vx	-	DCDA	-	-	-	-
89	Karpagam	28	5972	G2P1L1	Spont	-	-	32	Preterm	LSCS	Vx Vx	-	MCDA	-	-	-	-
90	Sathya	26	6172	G2P1L1	Spont	-	-	34	preterm	LSCS	Vx Breech	-	MCMA	P(cord entanglement)	-	-	-
91	Nasreema Banu	30	8026	G2P1L1	Spont	-	-	30	Preterm Prom	Vaginal	Vx Vx	-	MCDA	P	P	-	-
92	Begum John	30	14108	Primi	Induced(dru)	-	Severe preeclampsia	32	Preterm Prom	LSCS	Breech	-	MCDA	-	-	-	-
93	Janath	29	12102	Primi	Induced(dru)	P	GDM	36	-	LSCS	Breech Vx	-	DCDA	-	-	-	-
94	Malliga	24	12162	G2P2L2	Spont	-	anemia	33	Preterm	Vaginal	Vx Breech	-	MCDA	-	-	-	-
95	Indarani	26	17160	Primi	Spont	-	-	32	preterm	LSCS	Vx Vx	-	MCDA	-	-	-	-
96	Kamatchi	28	14320	Primi	Spont	-	-	37	-	Vaginal	Vx Breech	-	DCDA	-	-	-	-
97	Uma Rani	26	7008	Primi	Spont	-	-	38	-	Vaginal	Vx vx	P	DCDA	-	-	-	P
98	Kala	19	8521	Primi	Spont	-	-	36	-	LSCS	Vx Breech	-	MCDA	-	-	-	-
99	Indrani	30	12764	G2P1L1	Spont	-	-	34	-	LSCS	Breech Vx	-	DCDA	-	-	-	-
100	Selvi	27	11709	Primi	Induced(dru)	-	-	36	-	Vaginal	Vx Vx	-	DCDA	-	-	-	-

Dru - drugs, Vx- vertex, MCDA - monochorionic diamniotic, MCMA- monochorionic monoamniotic, GDM - Gestational diabetes mellitus, P -present